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The Cardiovascular Responses to a Cold and Warm Wind Stimulus

by

Aaron Tubman

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE

OF MASTER OF SCIENCE

Department of Physical Education & Sport Studies

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FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled The Cardiovascular Responses to a Cold and Warm Wind Stimulus submitted by Aaron Tubman in partial fulfilment of the requirements for the degree of MASTER OF SCIENCE.

[redacted]

DEDICATION

This thesis is dedicated to my Dad and Mom, Garth and Evelyn Tubman, whom I love with all my heart. It is they, who have given me faith in myself. Without their love and support I would never be where I am right now. Thanks for being the greatest parents a girl could ever ask for.

ABSTRACT

The cardiovascular responses to a cold (-6 to -8C) and warm (20 to 23C) wind stimulus, blown at 8 m/sec onto the face, were studied. The protocol utilized consisted of five continuous phases: rest, warm phase 1, cold phase, warm phase 2, and recovery. Two age-specific populations participated, 15 young subjects (19 to 30 years) and 7 old subjects (53 to 66 years). The physiological variables measured were heart rate, stroke volume, arterial blood pressure, and finger pulse wave amplitude. Cardiac output was determined from heart rate and stroke volume data.

The results indicate that the cold wind stimulus was sufficient to produce significant ($p<0.05$) changes from the rest conditions; including bradycardia, decreased pulse wave amplitude, and increased systolic and diastolic blood pressure, in both age groups. Both phases of the warm wind stimulus produced significant reductions in pulse wave amplitude. Stroke volume tended to increase in the cold phase, and cardiac output was maintained throughout the procedures. There were no significant differences between the young and old group for any of the cardiovascular variables measured.

The cardiovascular responses evoked by a cold wind have been associated with the diving reflex. The afferent pathway of the diving response is thought to be the trigeminal nerve, and the efferent pathway is primarily the vagus nerve. The results support this, since the components of the diving response occurred with only facial exposure, and because the immediate onset of bradycardia demonstrated indicates a vagal response. It appears that the diving response is temperature-dependent with the exception of the peripheral vasoconstriction which may be a 'wind reflex'.

The elicitation of the diving response with a cold wind has implications for individuals who experience angina pectoris. The results indicate that there can be increased pressure-volume work for the heart, due to the elevated arterial pressure. Therefore, increased myocardial oxygen demand evoked by a cold wind may be one of the mechanisms producing angina pectoris.

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1. INTRODUCTION

A. General Overview

The diving response refers to a sequence of physiological events including apnea, bradycardia, and redistribution of cardiac output, which are all under the control of multiple reflexes (Smith and Kampine, 1984). Cold stimulation of the receptors of the face initiates the diving reflex, inhibits the respiratory center (producing apnea), induces vagal bradycardia and general vasoconstriction (Daly and Angell-James, 1979). Arterial pressure is maintained due to an increase in peripheral resistance, while blood flow to the heart and brain is maintained through the absence of coronary and cerebral vasoconstriction. This response was first identified in seals and has been demonstrated to a lesser degree in humans.

The mediation of the bradycardia and associated responses of the diving response seems to originate from a multineuronal reflex acting through centers in the medulla oblongata. The cerebral cortex may not be essential for this response since decerebrate animals still exhibit the

response (Andersen, 1963; Whayne, Smith, Eger, Stoelting, and Whitcher, 1971; and Martner, Wadenvik, and Lisander, 1977). However Jones, Fisher, McTaggart, and West (1973) found that seals displayed an anticipatory bradycardia before a dive and cardioacceleration when approaching the surface which suggests that there can be modification of the diving response with higher brain center involvement. This occurrence has also been observed in man, where performance of a mental activity during the cold stimulus raised the threshold of the reflex response (Ross and Steptoe, 1980).

The afferent pathway of the diving reflex in diving and non-diving animals involves the trigeminal nerve as evidenced by abolishion of the response with trigeminal nerve lesions (Andersen, 1963; and Martner et al, 1977). Andersen (1963) has also shown that the greatest bradycardia response occurs when there is stimulation of the opthalamic branch. It has been hypothesized that the afferent pathway is similar for humans (Leblanc 1975; and Parfrey and Sheehan, 1975).

The efferent pathway of this multineuronal reflex seems to involve the sympathetic and vagal pathways; the vagus nerve being the most important. Administration of atropine, which blocks the vagal response, completely

abolishes bradycardia in ducks (Richet, 1899), porpoises (Irving, Scholander, and Grinnell, 1941), rabbits (Forster and Nyboer, 1955), seals (Murdaugh, Seabury, and Mitchell, 1961) and bullfrogs (Lillo, 1979) when immersed in cold water. However, beta-adrenergic blockade of the sympathetic response had little effect on bradycardia produced by immersion (Finley, Bonet, and Waxman, 1979). This research indicates the lesser involvement of the sympathetic nervous system in the production of the bradycardia response. However, an increase in sympathetic activity during facial immersion may be responsible for peripheral vasoconstriction rather than the bradycardia response (Finley et al, 1979; Hayward, Holmes, and Gooden, 1976). Finley et al (1979) found that atropine also inhibited the bradycardia response during face immersion in man suggesting similar mechanisms are involved for man and animal. However, the response in man is not as strong or fast in onset.

Bradycardia and reduced limb blood flow, characteristic of the diving response, have been demonstrated in several face immersion studies (Magel, McArdle, Weiss, Stone, and Newman, 1982; Whayne et al, 1977; Stromme, Kerem, and Elsner, 1970; Irving, 1963), however, few studies have utilized a cold wind stimulus

directed at the face. Hayward et al (1976), reported that a stream of cold air directed at the side of the face produced a 9% decrease in heart rate, 22% reduction in forearm blood flow, and a 36% increase in forearm vascular resistance.

Leblanc, Blais, Barabe, and Cote (1976), hypothesized that the lowering of heart rate during resting cold face bradycardia was due to stimulation of the vagal reflex through the trigeminal nerve by the cold wind. It was suggested by Leblanc, Cote, Dulac, and Dulong-Turcot (1978) that the cardiovascular response to a cold wind stimulus may be of importance in determining the etiology of angina pectoris. Angina pectoris is a condition which results from a transient, reversible myocardial ischemia and the chest pain is associated with a temporary reduction of cardiac output, stroke volume, and ventricular contractility (Smith and Kampine, 1984). Hattenhauer and Neill (1975) observed angina in four patients with coronary disease that breathed cold air through the nose, with no significant change in myocardial oxygen consumption or systolic blood pressure. In a similar study, Lassvik and Areskog (1979) reported a 7% decrement in exercise time with a cold environment and a lower maximal work capacity due to an increase in systolic blood pressure. Futhermore, Brown and Oldridge (1985) found

that exercise in a cold environment resulted in an earlier onset of angina than compared to a warm environment.

Two physiological mechanisms may be responsible for the elicitation of angina pectoris in the cold: the increased myocardial oxygen demand due to the increased pressure work of the heart, or the reflex vasoconstriction of the coronary arteries (Brown and Oldridge, 1985).

The literature suggests that an older population will exhibit less significant bradycardia and peripheral vasoconstriction when compared to a younger population. However, this difference may be due to lower fitness levels associated with aging producing a variation between the two age-specific populations.

B. Statement of the Problem

There is conflicting evidence in the literature, particularly in humans, of the appearance and magnitude of the major components of the diving response, and a lack of research concerning the stroke volume and cardiac output responses to a cold wind stimulus. The reason for the controversy may be due to differences in the temperature of the cold air stimulus as the response may be temperature-dependent (Whayne et al, 1977). The importance

of defining this cardiovascular response is that angina pectoris can be elicited by cold wind (Neill et al, 1974; Hattenhauer and Neill, 1975). Furthermore, it has been hypothesized that angina is evoked by an increase in myocardial oxygen consumption, directly related to increased pressure work as a result of elevated arterial blood pressure (Leon, Ammidi, and Leonard, 1969; Neill et al, 1974). Also, since most diving reflex studies have been conducted on young male subjects and controversy exists regarding age-related responses to cold stimuli, further research is necessary to define the etiology of the cardiovascular response of age-specific populations to different temperature gradients.

Therefore, the purpose of this study was to observe the effects of a wind stimulus on selected cardiovascular variables with male subjects of two different age groups. More specifically, to determine if there is any differences in the response due to different stimulus temperatures or due to age differences. The parameters of the cold stimulus were chosen to best simulate a natural winter environment.

2. REVIEW OF LITERATURE

A. Mechanisms of the Diving Reflex: Animal Studies

Andersen (1963) performed a study on intact and decerebrate ducks with and without various trigeminal lesions to determine the reflex nature of the diving response and its afferent pathway. He found that the respiratory and circulatory adjustments to water immersion of the head are medullary reflexes totally independent of higher levels of the central nervous system. Also, decerebrated ducks demonstrated bradycardia upon submersion, as well as, post-diving cardioacceleration upon surfacing. The circulatory and respiratory adjustments associated with the diving reflex have also been shown in cats and other mammals which are not habitual divers. Martner et al (1977) demonstrated that in the majority of "chronically" decerebrated cats, submersion of the head caused apnea and bradycardia with maintained arterial pressure providing evidence that this response is basically organized at the lower brainstem level.

The integration of the diving reflex may be subcortical since it is present in decorticated animals.

However, cortical influences are also capable of modifying the response (Wolf, 1964). Jones et al (1973) found that seals display an anticipatory bradycardia before a dive and can also increase their heart rate when approaching the surface. This research supports the involvement of higher brain centers.

Evidence indicating that the trigeminal nerve is the afferent pathway of the reflex arc was also provided by Andersen (1963). Surgical interruption of the major branches of the trigeminal nerve resulted in elimination of the reflex. More specifically, the opthalamic and mandibular divisions were found to be the most important elements in the afferent limb of the reflex arc. In a similar study, Dykes (1974) studied the effect of trigeminal neurotomy and local anesthesia upon bradycardia of immersion in seals. These animals still demonstrated bradycardia during immersion, however, the bradycardia was slower in onset compared to intact animals. It seems that the severing of one or two of the major pathways of the trigeminal nerve in the seal did not eliminate the immersion bradycardia which was observed in ducks.

The pattern of neural activity in the trigeminal nerve produced by immersion elicits the abrupt onset of bradycardia (Andersen, 1963; and Dykes, 1974). The slower

time course for bradycardia without this information, may be associated with simple apnea. For example, when two seals with anesthetized faces were forcefully respired under water no bradycardia was demonstrated (Dykes, 1974). This provides evidence that both trigeminal and respiratory stimuli normally elicit and maintain immersion bradycardia. Dykes (1974) suggested that there are two major sensory contributions necessary for the bradycardia observed during immersion: 1) the cessation of movements of the lungs and rib cage, and 2) a change in neural activity from receptors in the region of the face. Martner et al (1977) discovered a link between apnea and bradycardia, and hypothesized that the primary reflex excitation of the vagal cardioinhibitory fibers is elicited by water sensitive receptors. However, a secondary factor in production of bradycardia could be the direct pathways between respiratory and cardiovascular neuron pools in the brainstem or by reflexes from pulmonary and thoracic receptors.

The vagus and right phrenic nerves serve as the efferent pathway of the diving reflex. Vagotomized and atropinized ducks did not show bradycardia upon submersion in a study by Richet (1899). Irving et al (1941) demonstrated that atropine prevented the reflex bradycardia in the porpoise. Furthermore, Forster and Nyboer (1955)

found that the atropinized rabbit did not have the reflex bradycardia. Murdaugh et al (1961) showed that atropine prevented the reflex bradycardia in seals. Lillo (1979) found that bullfrogs demonstrated significant bradycardia upon submergence while the response was eliminated with vagal-block. This research supports the theory that normal reflex bradycardia is mediated via vagal stimulation.

The abrupt onset of bradycardia is thought to be due to an inhibition by the vagus nerve. This process is facilitated by decreased venous return due to closure of the sphincter in the inferior vena cava (Harrison and Kooyman, 1968; Harrison and Tomlinson, 1960; and Hol, Blix, and Myhre, 1975). Andersen (1963) summarized the afferent and efferent pathways of the diving reflex in the following way:

SENSORY RECEPTORS -- TRIGEMINAL NERVE -- MEDULLARY CENTERS -- VAGUS NERVE

B. Cardiovascular Response to Apnea and Face Immersion in Humans

It is still unknown if the mechanism producing bradycardia and decreased peripheral blood flow during apnea and face immersion in humans is the same as in diving mammals. The diving bradycardia is slower in humans compared to diving animals but occurs rapidly enough to be reflexly initiated. Research has shown that the diving reflex is mediated by the vagus nerve and is initiated by afferent signals from peripheral receptors in the face and nose in both humans and animals (Magel et al, 1982; Ross and Steptoe, 1980; and Stromme et al, 1970). The face immersion response in man is characterized by a rapid reduction in: heart rate (25%); skin blood flow (75%); and muscle blood flow (50%); and, an increase in arterial blood pressure (20%)(Smith and Kampine, 1984).

Studies on heart rate response to apnea and face immersion in humans have shown that apneic bradycardia is significantly greater during face immersion as compared to air (Craig, 1963; Elsner and Scholander, 1963; Irving, 1963; Magel et al, 1982; Ross and Steptoe, 1980; and Stromme et al, 1970). Irving (1963) demonstrated that the intervals between heart beats were greater with face

immersion when compared to air. Intervals between beats were lengthened to 1.1 seconds, whereas facial immersion produced intervals of approximately 1.4 to 1.6 seconds. Stromme et al (1970) observed a moderate bradycardia with subjects wearing a face mask and a greater bradycardia upon removal of the mask. This occurred because the area of skin covered was innervated by the opthalamic division of the trigeminal nerve. Parfrey and Sheehan (1975) simultaneously covered the nose, eyes, and mouth and observed a reduction in heart rate upon immersion which was not significantly different from that produced by breath-holding. Although irrigation of the nasal passages with cold water produces apnea and a rise in blood pressure, the diving response can also be elicited without water entering the nasal passages by subjects who wore a noseclip (Campbell, Gooden, and Lehman, 1969b).

The cardiovascular changes that occur with diving are dependent upon facial immersion and breath-holding (Campbell, Gooden, and Horowitz, 1969a). It seems that the cessation of breathing upon immersion is a result of conscious inhibition of the respiratory centers of the brain. Hamilton and Mayo (1944) demonstrated that reflex inhibition of breathing can occur when water touches the face. Thus, it appears that breath-holding involves several

physiological events which initiate the diving response: intrathoracic pressure and its effect on venous return, blood gas changes, the absence of rhythmical stimulation of pulmonary stretch receptors, and the direct effect of inhibition of the respiratory center on the cardiovascular neurons in the brainstem (Elsner and Gooden, 1983).

There is controversy in the literature concerning the effect of variations in intrathoracic pressure and changes in venous return on the diving response. Intrathoracic pressure varies with the lung volume prior to and during breath-holding. As intrathoracic pressure becomes more negative in proportion to the depth of inspiration, diastolic filling and venous return are facilitated which results in an increased stroke volume and a rise in systolic arterial pressure (Magel et al, 1982). These events would stimulate the sinoaortic baroreceptors causing a reflex slowing of the heart rate (Craig, 1963; and Magel et al, 1982). Craig (1965) showed that bradycardia was greatest following maximal inspiration than after normal inspiration. However, Kawakami, Natelson, and DuBois (1967) saw no change in the magnitude of bradycardia during face immersion with apnea at the end of expiration or full inspiration.

The role of chemoreceptors in producing bradycardia

has been investigated. It has been proposed that diving bradycardia occurs in man before any major changes could occur in arterial oxygen or carbon dioxide tensions (Olsen, Fanestil, and Scholander, 1962a). Inhalation of a hypercapnic gas mixture did not show any differences in heart rate response in comparison to breathing air in a study by Kawakami et al (1967). Also, Elsner, Gooden, and Robinson (1971a) reported that arterial hypercapnia did not elicit diving bradycardia indicating that arterial carbon dioxide levels are not of great importance in the production of the diving response. However, some studies (Moore, Elsner, Lally, and Hong, 1972b) have demonstrated that the alveolar tension of oxygen may modify the bradycardia reflex. The bradycardia response was enhanced in the presence of low alveolar oxygen tension during both rest and exercise, but appeared to be independent of alveolar carbon dioxide tension (Moore et al, 1972b). Therefore, it has been suggested that arterial oxygen tension, can modify the intensity of the diving response through the peripheral chemoreceptors (Elsner and Gooden, 1983).

The administration of various types of drugs have allowed researchers to investigate the origin of the diving response in humans. Experiments utilizing atropine

demonstrate blockage or reduction of the bradycardia reflex indicating the important role of the vagal nerve in the development of the diving response (Heistad, Abboud, and Eckstein, 1968; Finley et al, 1979). In conjunction with these conclusions, experiments utilizing alpha or beta sympathetic blockade during face immersion, did not affect the development of bradycardia (Finley et al, 1968). This suggests that hypertension produced by the sympathetic response is not essential in the development of the diving response whereas the vagal influence is necessary. The carotid sinus reflex does not initiate the bradycardia, however, it may have a role in maintaining it for a short duration.

General anesthesia has been shown to decrease the bradycardial response from 29% to 3.6% below the control rate during a cold application to the face and simultaneous breath-holding (Whayne et al, 1971). It was thought that anesthesia reduced the parasympathetic response and enhanced the sympathetic component.

The magnitude of the bradycardia response in man may be dependent on the temperature of the stimulus. The bradycardia response to breath-hold diving in water at 22C was greater than at 29.5C and 34.5C (Craig, 1963). Similar results were found by Whayne and Killip (1967). The

augmentation of reflex bradycardia at lower water temperatures has been confirmed by several investigators (Kawakami et al, 1967; Moore et al, 1972a; Parfrey and Sheehan, 1975), however, it has been proposed that sensory modalities other than temperature are responsible since significant bradycardia and reduction in peripheral blood flow has been shown with immersion in 34C water (Campbell et al, 1969b). Since water at this temperature feels neither warm nor cold it may be the sensation of wetness that elicits the response.

Face immersion studies indicate that the initiation of the diving response in man is due to stimulation of the receptors of the face. The reflex inhibits the respiratory center producing apnea, as well as, eliciting vagal bradycardia and general vasoconstriction (Hamilton and Mayo, 1944; and Daly et al, 1979). During apnea, there is a reduced afferent input from the pulmonary stretch receptors and a cessation of the central inspiratory neuronal activity, allowing expression of the chemoreceptor vagal effects (Elsner et al, 1983; and Smith and Kampine, 1984). As a result, there is a continuation and reinforcement of the existing bradycardia and vasoconstriction. Arterial blood pressure is maintained or increased despite the reflex bradycardia, and is due to the extensive

vasoconstriction causing large increases in peripheral resistance (Irving, 1963; Stromme et al, 1970; and Elsner and Gooden, 1983). Studies utilizing sympathetic and parasympathetic blockades have shown that the pathways for the diving response in man are similar to those in animals. However some differences exist as there is evidence that the human response is temperature-dependent, whereas stimulus temperature does not affect the reflex in animals.

C. Cardiovascular Response in Humans to Facial Cooling

Very few studies have utilized cold wind blowing on the face as the cold stimulus to evoke the diving reflex. Hayward et al (1976) reported that a stream cold air (0C) directed at the side of the face produced a 9% decrease in heart rate, a 22% reduction in forearm blood flow, and a 36% increase in forearm vascular resistance, however cold air directed at the front of the face did not produce a significant bradycardia (6%). Leblanc et al (1978) used a 0C wind at a velocity of 18.3 m/sec as the cold face stimulus. Both subject groups, young (20-40) and old (53-60), demonstrated significant bradycardia. Riggs, Johnson, Konopka, and Kilgour (1981) performed a study

using a wind velocity of 6.5 m/sec with two different cold wind temperatures (2C and 10C) and subjects demonstrated significant bradycardia with both wind temperatures during exercise. It appears that the cold receptors involved in the bradycardia response with facial cooling, are sensitive to temperatures 10C and lower.

It appears that the changes in facial temperature may be related to the reflex lowering of heart rate. A linear relationship has been reported between heart rate and skin temperature between 35C and 23C (LeBlanc, 1975). It was suggested by Riggs et al (1981) that the bradycardia response to facial cooling during exercise is mediated through a central thermoregulatory response. Leblanc et al (1976), hypothesized that the lowering of heart rate during facial cooling was due to stimulation of a vagal reflex associated with stimulation of the trigeminal nerve by the cold wind.

New interest has recently focussed on the problem of angina pectoris elicited by a cold wind. Leblanc, Dulac, Cote, and Girard (1975) suggest the pain of angina pectoris produced when the face is exposed to cold wind may be related to reflex bradycardia. However, it has been suggested by other authors that during cold exposure the increase in peripheral resistance, produces elevated

arterial blood pressure, resulting in increased myocardial oxygen consumption (Neill et al, 1974). In Neill's study, the cold stimulus also provoked angina pectoris in 5 patients and myocardial hypoxia, shown by changes in coronary venous blood, in 7 patients out of 25 patients. Contrary to most studies, Leon et al (1969) found that although cold air (-16C) breathing in normal men elicited an increase in stroke volume there was no change in heart rate or peripheral resistance. Yet, exposure to a cold environment evoked an increase in peripheral resistance without changing heart rate. Both conditions elicited an increase in myocardial oxygen requirements which Leon et al (1969) hypothesized would precipitate angina in a subject with sufficient ischemic heart disease. These studies provide evidence that angina pectoris is elicited due to an increased myocardial oxygen demand, as a result of increased pressure work of the heart.

However, it has also been hypothesized that cold exposure does not increase the myocardial oxygen demand, but instead decreases the oxygen supply to the heart by reflex constriction of the coronary arteries (Brown and Oldridge, 1985: and Hattenhauer and Neill, 1975). In cold air inspiration it has been suggested that cold receptors, located in the mucous membranes of the airways are

stimulated by the cold air producing a reflex coronary artery vasoconstriction. This theory is supported by Hattenhauer and Neill (1975) who found no significant changes in myocardial oxygen consumption or in systemic arterial blood pressure, when subjects breathed cold air (-20C) through the nose. Yet, 4 out of the 17 patients developed angina pectoris. The results of studies concerning the effect of cold exposure on coronary vascular resistance, however, are conflicting. Berne (1953) showed that ice water immersion of open-chested anesthetized dogs failed to produce reflex coronary vasoconstriction. Yet Gilbert, Fenn, and LeRoy (1941) demonstrated reflex coronary vasoconstriction on decerebrate dogs when ice water was applied to the nasal mucous membranes.

Studies showing cold stimulus on the face show conflicting heart rate, systemic arterial pressure, and peripheral resistance responses; and a lack of research concerning stroke volume and cardiac output responses. It has been hypothesized that the diving reflex can be evoked by a cold wind on the face through stimulation of the trigeminal nerve and the resulting vagal reflex (Le Blanc et al, 1976). This may be important in determining the etiology of angina pectoris, which is thought to be due to an increased myocardial oxygen demand, as a result of

cardiovascular responses occurring due to facial cooling. More research is required to fully identify the cardiovascular responses elicited by a cold wind in normal and patient populations.

D. The Effects of Age on the Diving Reflex

Age may be a factor modifying the magnitude of the diving response. It has been reported (Hong, Blix, and Myhre, 1970) that bradycardia becomes less pronounced with advancing age. Similar findings were reported by Gooden, Holdstock, and Hampton (1978). They found a significant correlation between the bradycardia produced by face immersion breath-holding and the subjects' age. Contrary to these results, Leblanc et al (1978) found a more pronounced bradycardia was demonstrated in older subjects (53 to 60 years) than in younger subjects (20 to 40 years) during the cold face test. Thermoregulatory studies (Wagner, Robinson, and Marino, 1974) indicate that aging may be accompanied by a reduction in functional, if not, anatomical cutaneous vascularization. During cold exposure, older men tend to have higher finger blood flow than a younger age group indicating a decrease in peripheral vascular reactivity to

cold stress (Wagner et al, 1974) and therefore, a reduced ability for peripheral vasoconstriction in response to cold.

A difference in the diving response as demonstrated by older subjects may be related to the often associated lower fitness level of this group compared with younger subjects. It is well documented that there is a cardiac adaptation with prolonged periods of physical training. The resting bradycardia produced in trained individuals, is explained by the cholinergic dominance concept (Stromme et al, 1970). It has been hypothesized that trained subjects would elicit a more significant bradycardia during apneic face immersion than physically unfit persons since vagal impulses have been proposed to be responsible for diving bradycardia (Stromme et al, 1970). Bove, Lynch, Connell, and Harding (1968) reported that a 10 week physical training program augmented the diving bradycardia. However, Whayne and Killip (1967) reported that heart rate responses to apnea and face immersion were similar in trained and untrained subjects. Stromme et al (1970) also found no consistent relationship between the degree of bradycardia during face immersion and the level of physical fitness.

Although few studies have been performed to determine if the magnitude of the diving response is different in an

older population, the available evidence shows a reduced bradycardia and a reduced peripheral resistance as compared to a younger population. This difference may be in part due to a lower fitness level associated with aging.

E. Validation of Impedance Cardiography

Impedance cardiography is a non-invasive technique for measuring cardiac output. This technique is dependent on the detection of changes in transthoracic electrical impedance associated with the cardiac cycle. Validation of the impedance cardiography method has been provided by several investigators (Edmunds, Godfrey, and Tooley, 1982; Miles, Sawka, Wilde, Doerr, Frey, and Glaser, 1981; Gabriel, Atterhog, Oro, and Ekelund, 1976; and Denniston, Mah, Reeves, Cruz, Cymerman, and Grover, 1976) and there is evidence that this technique is an acceptable measure of cardiac output at rest and at submaximal exercise. Kubicek et al (1974), reported that in the absence of distorted dZ/dt waveforms, valvular shunts, and absence of left and right shunts, it appears that the impedance cardiograph produces cardiac output values approximately equal to the dye dilution or Fick methods (Edmunds et al, 1982). In a

study completed by Teo et al (1985) and Hetherington et al (1985), a highly significant correlation was found between the measurements made by impedance cardiography and the Fick technique. They also found no significant systematic error in the impedance method when compared to the direct Fick method (Teo et al, 1985; Hetherington et al, 1985). The impedance cardiography technique is a safe, convenient, and valid method of measuring cardiac output under rest conditions.

3. METHODS AND PROCEDURES

A. Subjects

The subjects were two groups of healthy, sedentary male subjects between the ages of 19-30 years (N=15) and 53-66 years (N=7) who signed an informed consent and agreed to participate as subjects (Appendix 1). The subjects had a mean resting heart rate of 73 beats per minute and range of 68 to 86 beats per minute. Subjects were required to fast for 4 hours prior to the experimental trials, and were requested to abstain from alcohol or prescription drugs 12 hours prior to the testing. All tests were carried out at approximately the same time of day.

	HEIGHT (cm)		WEIGHT(kg)	
SUBJECT GROUP	MEAN	S.D.	MEAN	S.D.
OLD	179.8	8.2	74.4	9.1
YOUNG	179.3	14.3	82.5	10.3

Table 1: Physical description of old and young subject groups.

B. Procedures

Preparation for each subject involved placement of three circular, adhesive electrodes in right arm (RA), left arm (LA), and right leg (RL) positions for the electrocardiogram; and placement of four bands of adhesive, mylar-backed aluminum electrodes, two around the neck and two around the abdomen for the measurement of stroke volume and cardiac output (Figure 1). The two neck bands were placed 3-5 cm apart, while one trunk electrode was placed inferior to the xiphoid process and the other 3-5 cm below at a level superior to the umbilicus. The two outer electrodes transmitted a constant sinusoidal alternating current (4 mA r.m.s. at 100 KHz) through the thorax and the changes in transthoracic electric impedance were detected by the two inner electrodes by the impedance cardiograph (Kubicek et al, 1966 and 1974). A blood pressure cuff was placed on the left arm of the subject with the lower edge approximately 2.5 inches above the antecubital space. The right thumb was fitted in a strain-gauge apparatus with the forearm supported by a table. All equipment and readings were checked prior to each experiment. The subjects were requested to relax and remain as still as possible, and to avoid sighing or yawning throughout the procedures. The

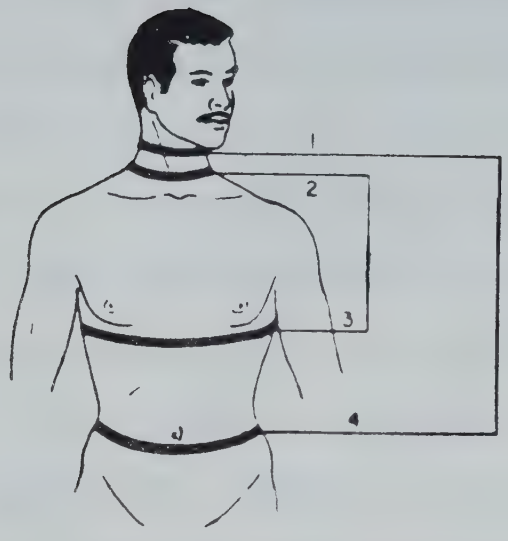

Figure 1: Electrode placement for the measurement of cardiac output by means of electrical impedance. Electrodes 1 and 4 received a constant, sinusoidal current (4 mA, 100 kHz) and the voltage representing impedance changes was detected by electrodes 2 and 3 (modified from Denniston, J.C., J.T. Maher, J.T. Reeves, J.C. Cruz, A. Cymerman, and R.F. Grover. Measurement of CO by electrical impedance at rest and during exercise. J. Appl. Physiol. 40:91-95, 1976).

subjects were required to rest for 5 minutes or until the resting heart rate stabilized. The experimental procedures consisted of five, two minute continuous phases: phase 1 = rest, phase 2 = warm wind, phase 3 = cold wind, phase 4 = warm wind, and phase 5 = recovery. Wind velocity for all phases was 8 m/sec. The subjects were covered with towels leaving only the face exposed to the wind.

During phase 1, the subject was seated comfortably with his face forward against the facebox opening with no wind stimulus. Phase 2 involved a warm wind (21 to 23C) blown on the face. During phase 3 a cold wind (-6 to -8C) was blown on the face, followed by a warm wind in phase 4. Finally, there was no stimulus in phase 5. Each wind phase consisted of 3 stages: immediate start, one-minute, and two-minute. The rest and recovery phases included only two stages: the one-minute and two-minute. The protocol was represented by these stages:

Stage 1 = One-Minute Rest

Stage 2 = Two-Minute Rest

Stage 3 = Start Warm Wind Stimulus Phase 1

Stage 4 = One-Minute Warm Phase 1

Stage 5 = Two-Minute Warm Phase 1

Stage 6 = Start Cold Wind Stimulus Phase

Stage 7 = One-Minute Cold Phase

Stage 8 = Two-Minute Cold Phase

Stage 9 = Start Warm Wind Stimulus Phase 2

Stage 10= One-Minute Warm Phase 2

Stage 11= Two-Minute Warm Phase 2

Stage 12= One-Minute Recovery Phase

Stage 13= Two-Minute Recovery Phase

Measurement of heart rate, stroke volume, and finger pulse wave heights were taken at all stages. Cardiac output values were calculated from heart rate and stroke volume measurements. Systolic blood pressure and diastolic blood pressure measurements were recorded only at the one-minute stage of each phase. Arterial blood pressure measurements were made in only 11 of the young group's tests and in all the older group's tests. The room temperature was maintained at approximately 23C throughout all procedures.

C. Apparatus and Techniques

The wind stimulus apparatus consisted of two channels, one an open channel for the flow of room air, and the other channel containing a small radiator which cooled the air as

it passed through to the facebox exit duct (Figure 2). Room air was propelled through the face-box by a centrifugal fan (Fasco Ind. Inc., Roch N.Y.; Blower #U50755-J296 MTR #61R3AC) at the end of the unit. A sliding damper was used to control the air flow through either channel. The two temperature channels merged into a cone-shaped exit duct with a screen acting as a division for the beginning of the exit duct. The wind stimulus flowed through a 10 by 10 cm opening at the end of the exit duct. The top of the square opening had a foam edging for the subjects' forehead. A large cooling tank filled with a glycol-water mixture formed the lower portion of the apparatus. The tank contained cooling coils from a refrigeration unit which maintained the glycol-water mixture at -10C. This fluid was further cooled to -16C at testing times by the addition of dry ice. The cold fluid was circulated up through the heat exchanger in the facebox and back into the cooling tank in a continuous cycle with a small, centrifugal water pump (Cole-Parmer Instrument Co., Chicago, Model 60648). The temperature of the air stream was monitored with a thermocouple attached in the facebox exit duct approximately 5 cm in front of the opening. The velocity of the wind was measured by a vane anemometer (West Germany, Model 1400/401315,).

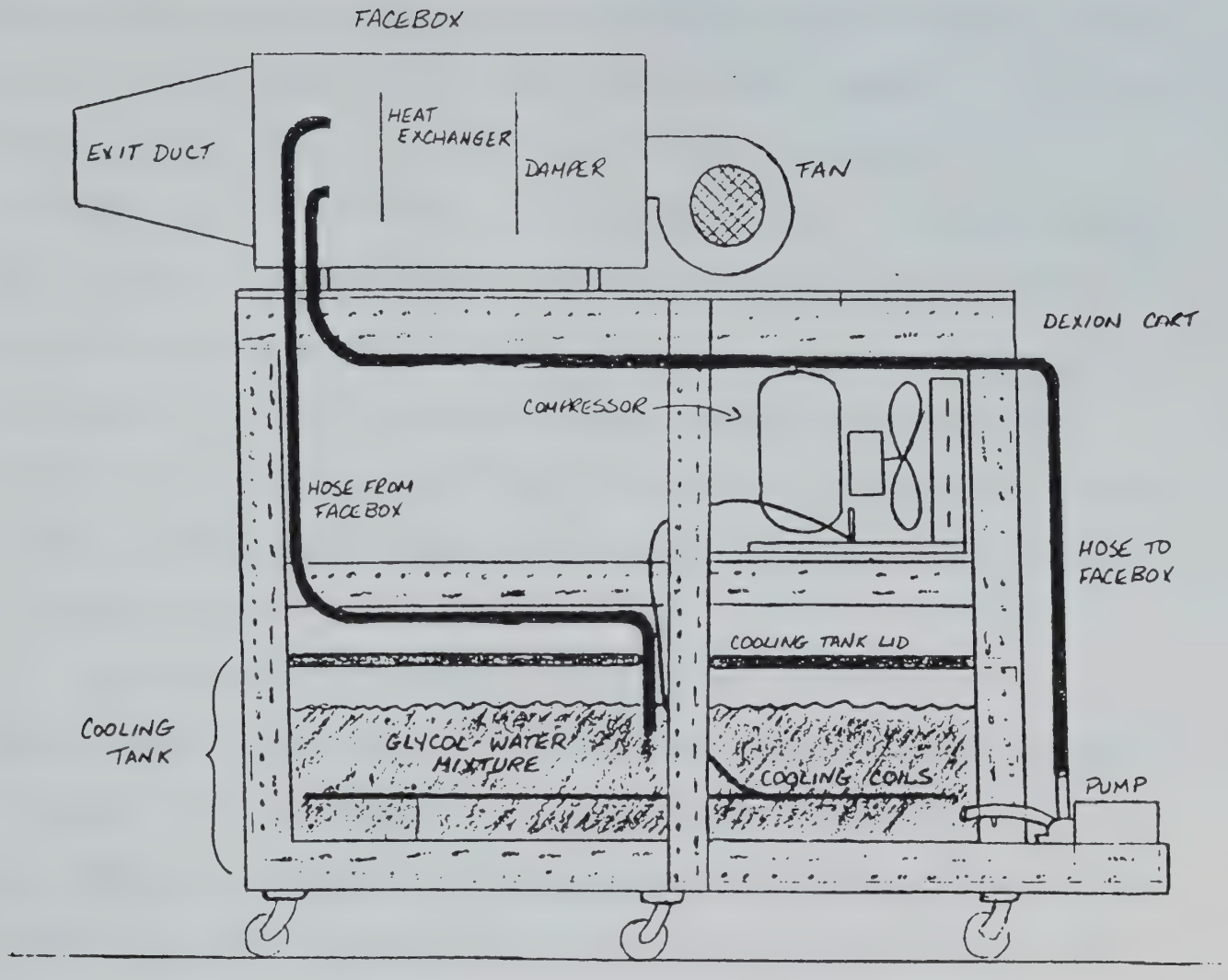

Figure 2: Diagram of wind stimulus and cooling tank apparatus utilized in experimental procedures (modified from drawing by Dr. W. Cottle, University of Alberta).

Heart rate was monitored on a Biotach mode channel of the electrocardiograph and recorded on an polygraph (Gould Inc., Cleveland, Model 24005) at a chart speed of 2 mm/sec. Heart rates were counted in continuous periods of 10 seconds and converted to beats per minute. A second channel was used to record finger pulse waves at 2 mm/sec with a strain-gauge plethysmograph (Vasculab, Model SPG16). The amplitude of the pulse waves was measured to estimate qualitative changes in finger blood flow. Brachial arterial blood pressure was measured manually by a baumamometer (Standby Model).

Calculation of cardiac output was derived from measurements recorded on a Minnesota Impedance Cardiograph (Surcom Inc., Minnesota, Model 304B). Changes in transthoracic electric impedance were detected between the inner neck and chest electrodes and the total impedance (Zo) was computed and displayed on the Impedance Cardiograph. An electrocardiograph, phonocardiograph, and the rate of change of impedance through each phase of the cardiac cycle (dZ/dt) were simultaneously recorded on a polygraph (Gould, Model No. 8188-4400-00) at a chart speed of 50 mm/sec. The electrocardiograph and heart sounds, detected by a phonomicrophone (Hewlett Packard, Model 21050A: Contact Sensor), were amplified by a D.C. Amplifier (Gould, Model 2512549).

Calculations of stroke volume and cardiac output were made using the following expression:

$$\text{Stroke Volume} = \frac{P \times L^2 \times T \times dZ/dtmin}{Zo^2}$$

where, P = resistivity of blood at body temperature as given by $53.2e^{0.022H}$,

H = hematocrit (%),

L = average distance between the inner pair of electrodes measured at the anterior and posterior midline (cm).

T = left ventricular ejection time (sec),

dZ/dtmin = minimum value for the rate of change of impedance occurring during the cardiac cycle (Ohms/sec), and

Zo = total impedance (Ohms) was obtained as described above.

Ejection times were measured using the recording of dZ/dt and the phonocardiograph. Recordings were made over 6 to 8 cardiac cycles and the calculations were based on 5 cardiac cycles. The hematocrit values were measured from a blood sample obtained by using a finger prick method immediately following the experimental procedures.

D. Data Analysis

Statistical analysis of the heart rate, stroke volume, cardiac output, and finger pulse wave amplitude consisted of the Bonferroni multiple T-tests. Systolic and diastolic blood pressures were analyzed with a two-way analysis of variance with repeated measures from each stage, followed with the Duncan post-hoc test. Statistical significance was accepted at the .05 level in all cases.

4. RESULTS

The mean data for all the variables measured are graphically represented. The graphs are of two types: the mean combined data of both subject groups, and the mean data for the individual groups (young and old). The abscissa in each graph represents the variable measured, the origin in each graph represents the sequence of the experimental stages.

A. Heart Rate

Mean heart rate was significantly decreased from resting conditions at the beginning of the cold phase and warm phase 2 (Figure 3). This bradycardia occurred immediately at the first stage of each of these phases, and then heart rate began to increase in the final two stages of these phases. There was a significant heart rate change in the group from a resting value of 73 beats/min to 67 beats/min (8.2%) at the onset of the cold stimulus. Heart rate increased in the final two stages of the cold phase,

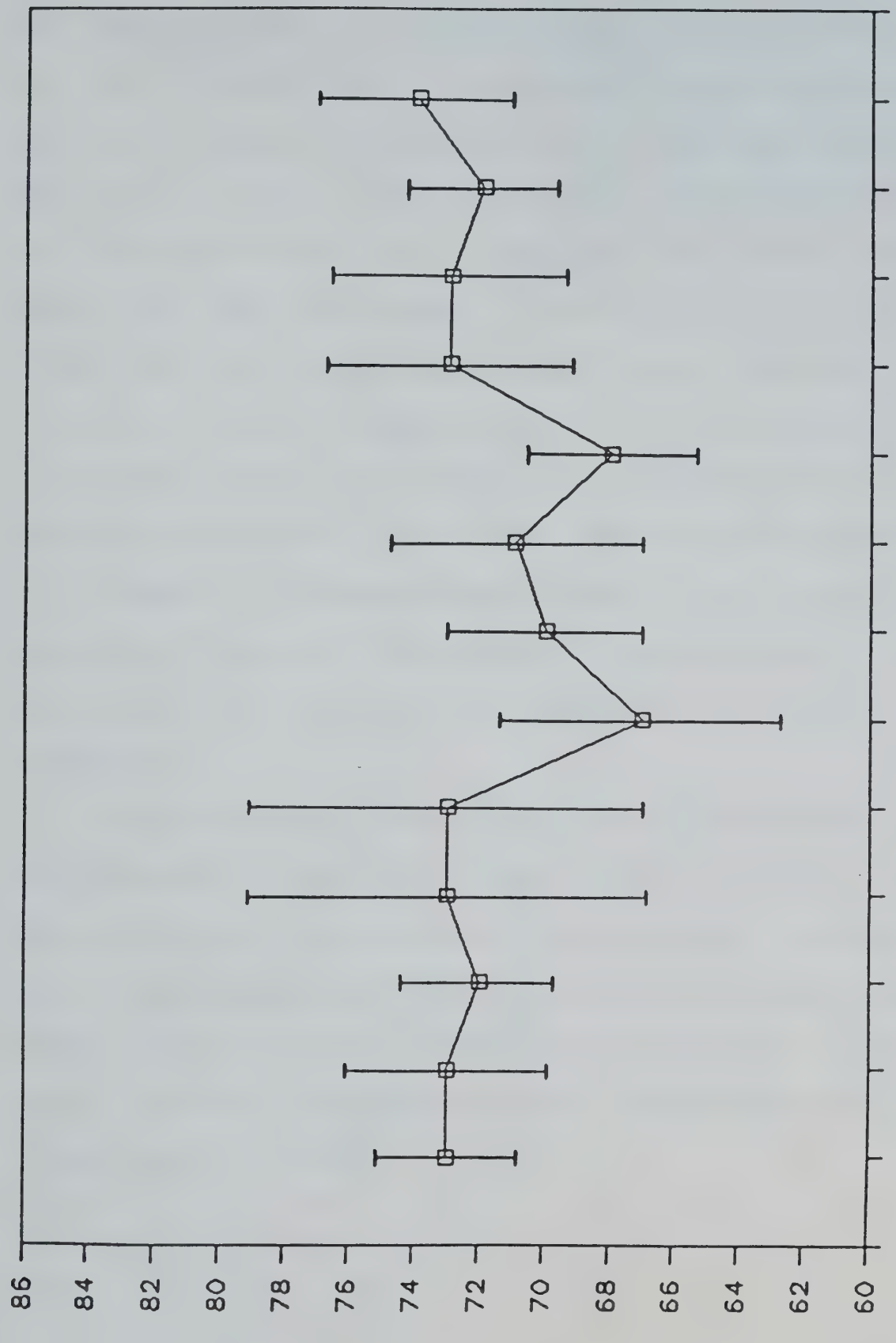

Figure 3: Mean heart rate responses of both subject groups during the experimental stages.

but did not return to resting values. A significant decrease in heart rate from resting values was observed in the first stage of the warm phase 2, although this result was not as large as during the cold phase. With the onset of the second warm wind stimulus the mean heart rate for both young and old dropped to 68 beats/min ($p<0.05$), 6.8% lower than the resting heart rate values. However, in the final two stages of phase 2 the heart rate returned rapidly to resting values. The young group tended to have an increase in recovery heart rate above resting values. Heart rate tended to decrease from control values within 0 to 10 seconds of the cold wind stimulus. Lengthening of the first or second R-R intervals was evident for 86% of the subjects.

The mean heart rate values of the young group were significantly higher than those of the old group throughout the procedures. Both subject groups showed a similar heart rate response pattern (Figure 4). However, the younger group showed a stronger bradycardia response than the older group, but the difference between the groups was not significant.

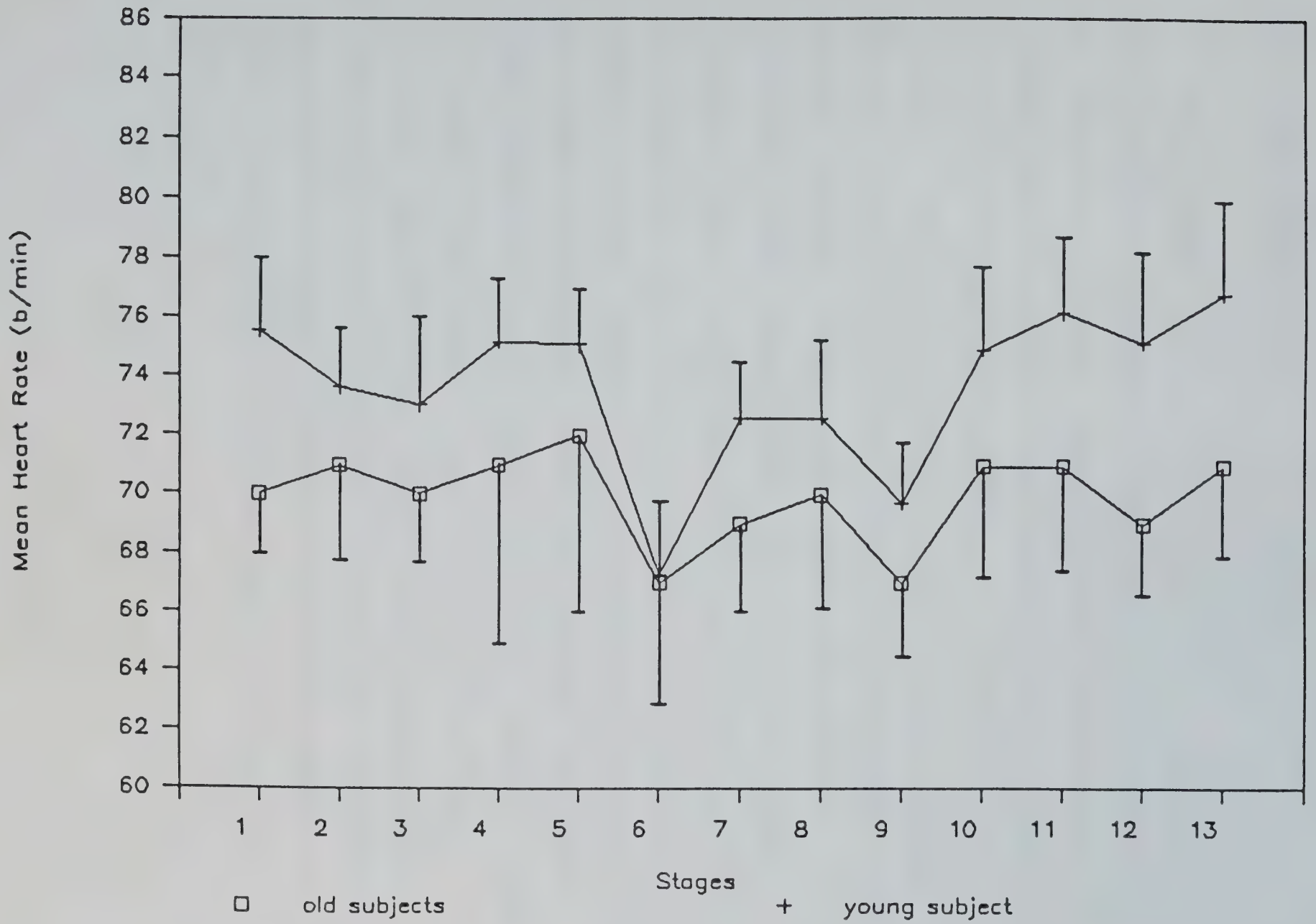

Figure 4: Mean heart rate responses of the young (+) and old (□) subject groups during the experimental stages.

B. Stroke Volume

Mean resting stroke volume values for the groups were not significantly different from stroke volume values for any of the wind stimulus phases. However, mean stroke volume tended to increase from resting values at stage 1 of both the cold phase and warm phase 2, as is evident in Figure 5. The onset of the cold wind stimulus increased stroke volume to 90.3 ml/beat from a resting stroke volume of 86.6 ml/beat, and the second warm wind stimulus increased stroke volume to 90.7 ml/beat for the groups. Although, these changes were not statistically significant from resting values, they were significantly higher than recovery values. The mean stroke volume values of the older group were significantly lower than those of the younger group throughout the procedure (Figure 6). However, changes in stroke volume between groups were not statistically different over the stages. The increase in stroke volume from rest at the onset of the cold phase and warm phase 2, appeared to be greater in the young group as compared to the old group but this difference was not statistically significant .

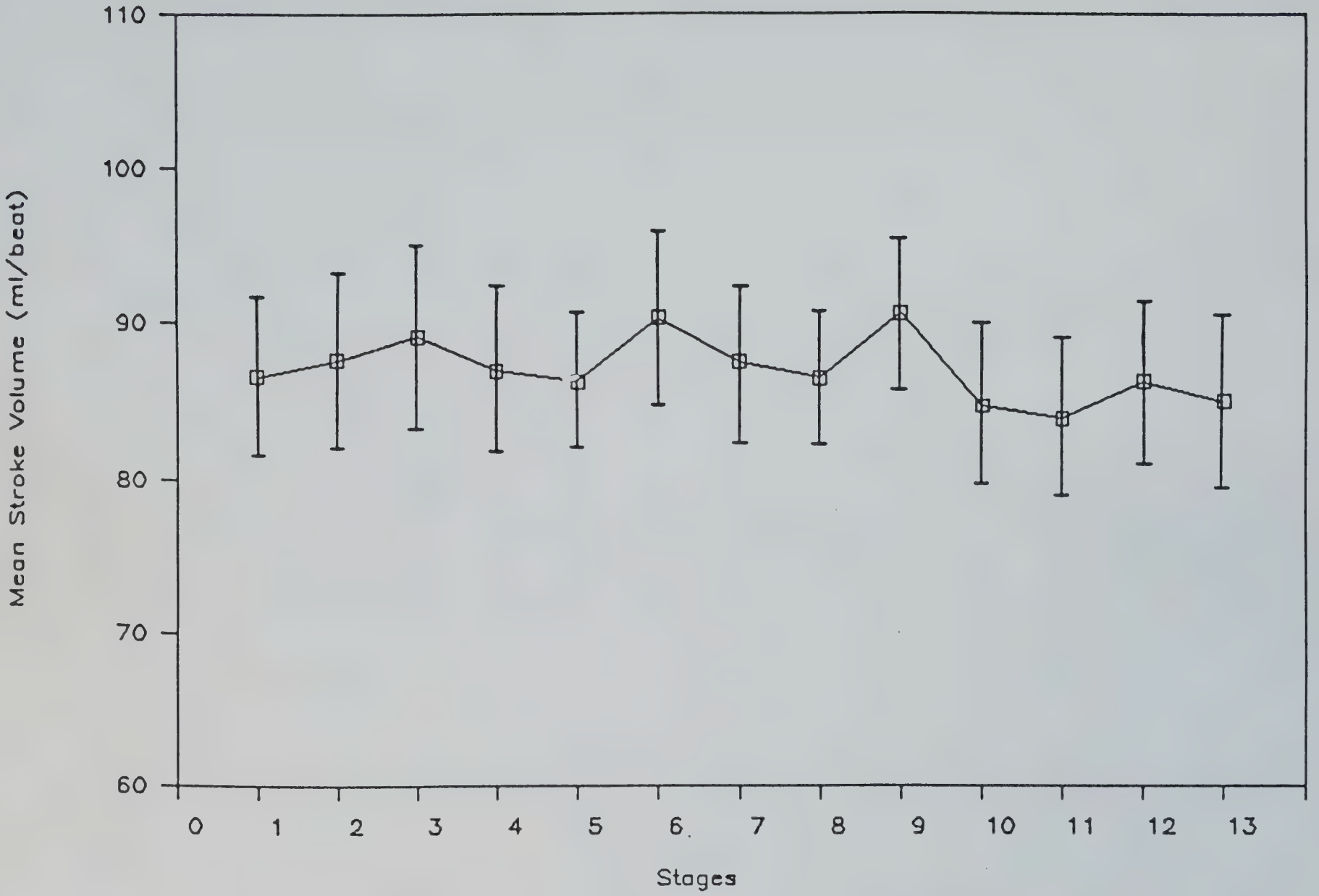

Figure 5: Mean stroke volume responses of both subject groups during the experimental stages.

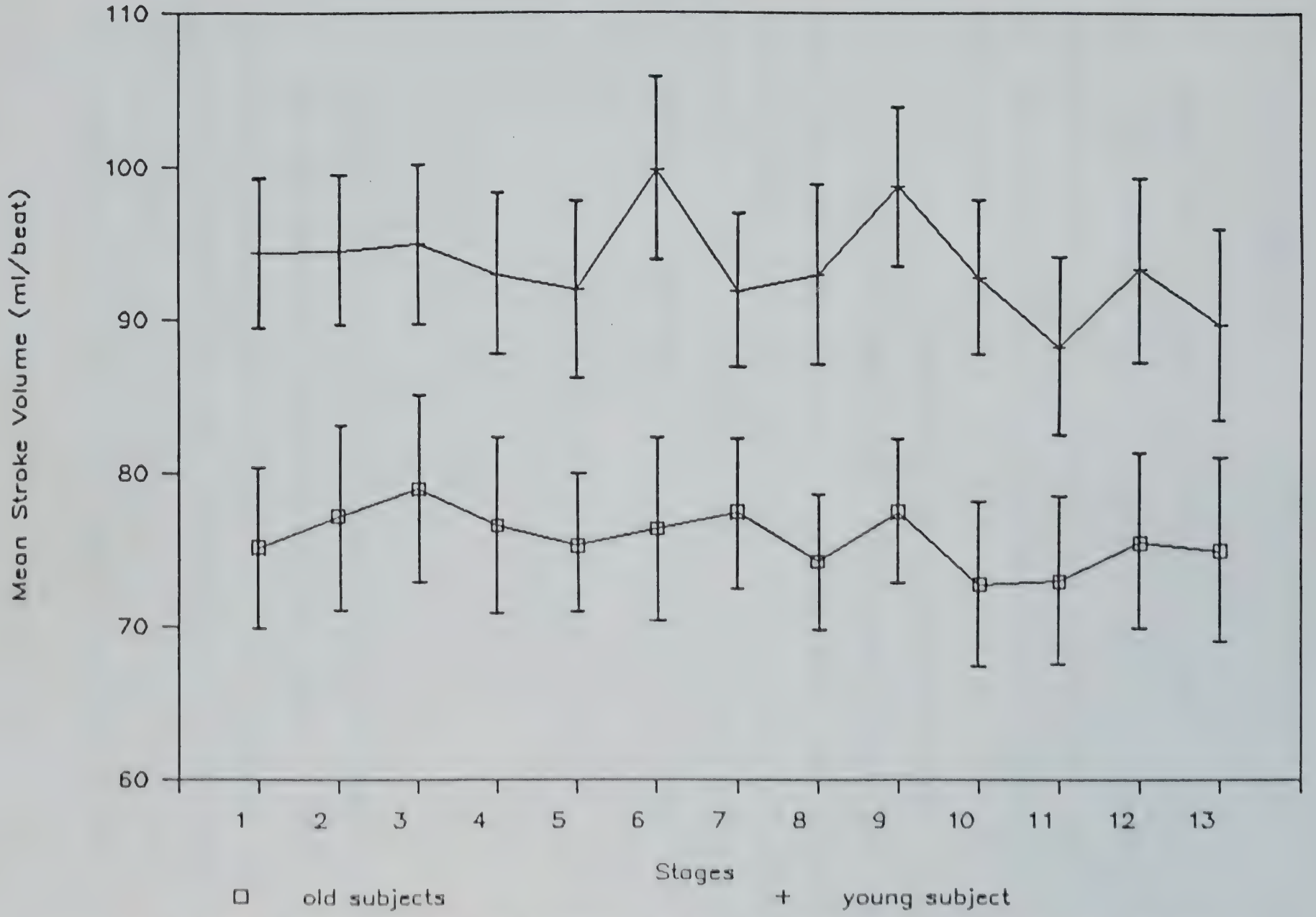

Figure 6: Mean stroke volume responses of the young (+) and old (□) subject groups during the experimental stages.

C. Cardiac Output

There was no significant change in cardiac output in any of the phases (Figure 7). Cardiac output values did decrease slightly from resting levels at the beginning of the cold phase, however, this change was not statistically significant.

Descriptively, the two groups showed equivalent cardiac output responses at the cold phase (Figure 8). The young subjects demonstrated a decrease from a mean resting cardiac output value of 7.23 l/min to 6.86 l/min (14.6%) at the start of the cold wind stimulus, and the old group only demonstrated a decrease from 5.47 l/min to 5.12 l/min (6.4%).

D. Finger Pulse Wave Amplitude

There were significant changes in the mean amplitude of the pulse waves of the groups at each phase of wind stimulus (Figure 9). The decreases in the mean pulse wave amplitude at the first stage of warm phase 1, cold phase, and warm phase 2; were different from rest values ($p<0.05$). The pulse wave response in the final two stages of the warm

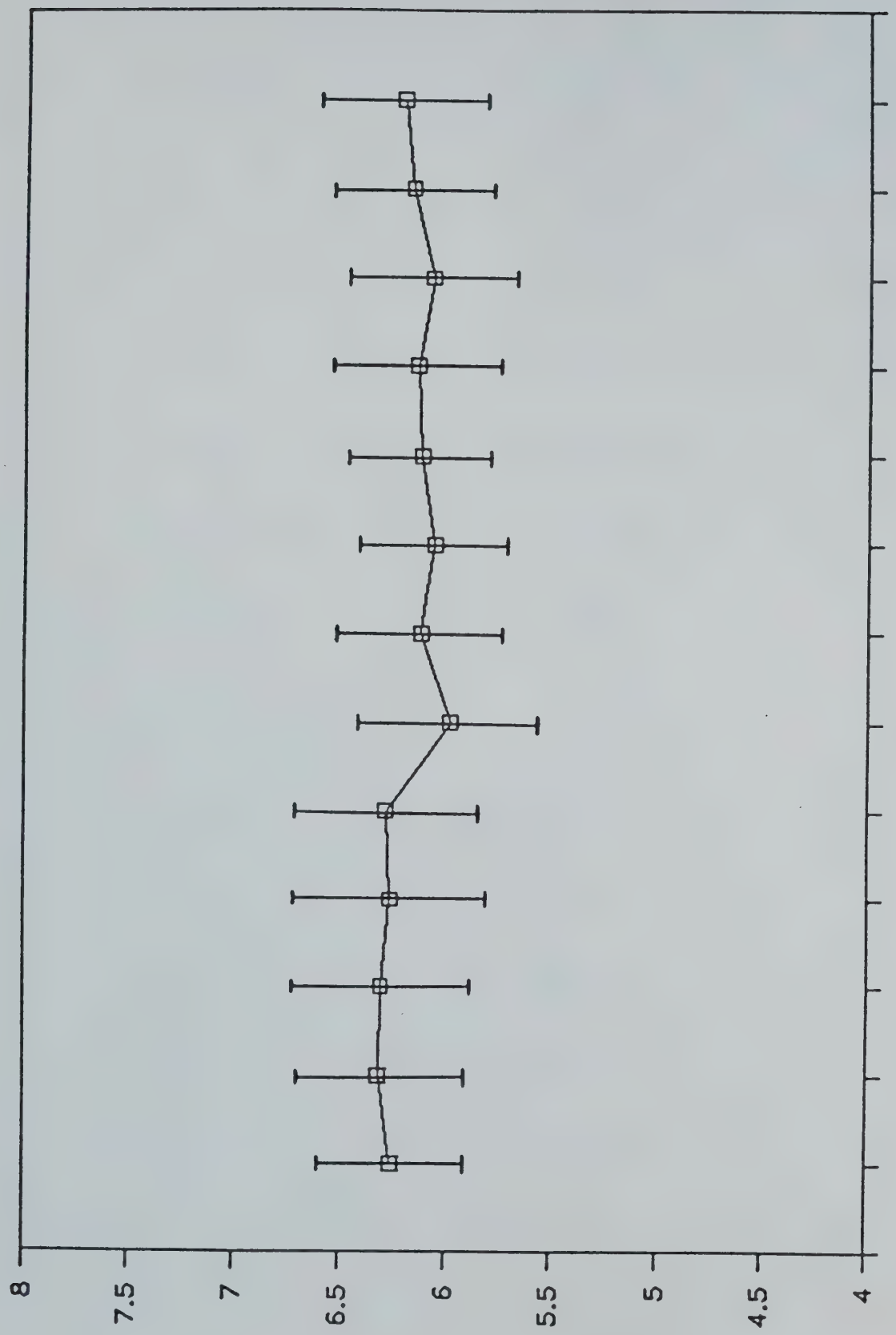

Figure 7: Mean cardiac output responses of both subject groups during the experimental stages.

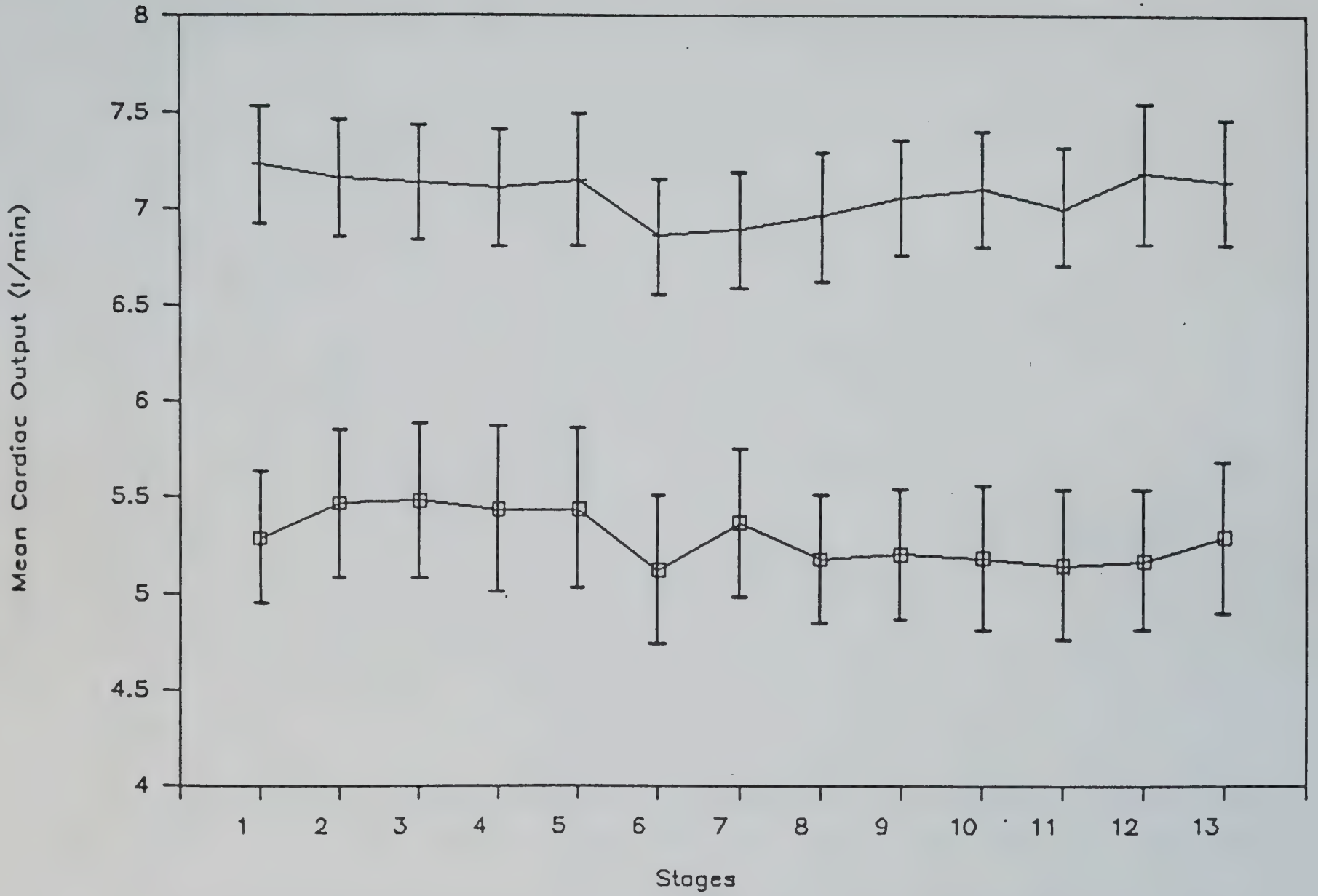

Figure 8: Mean cardiac output responses of the young (+) and old (□) subject groups during the experimental stages.

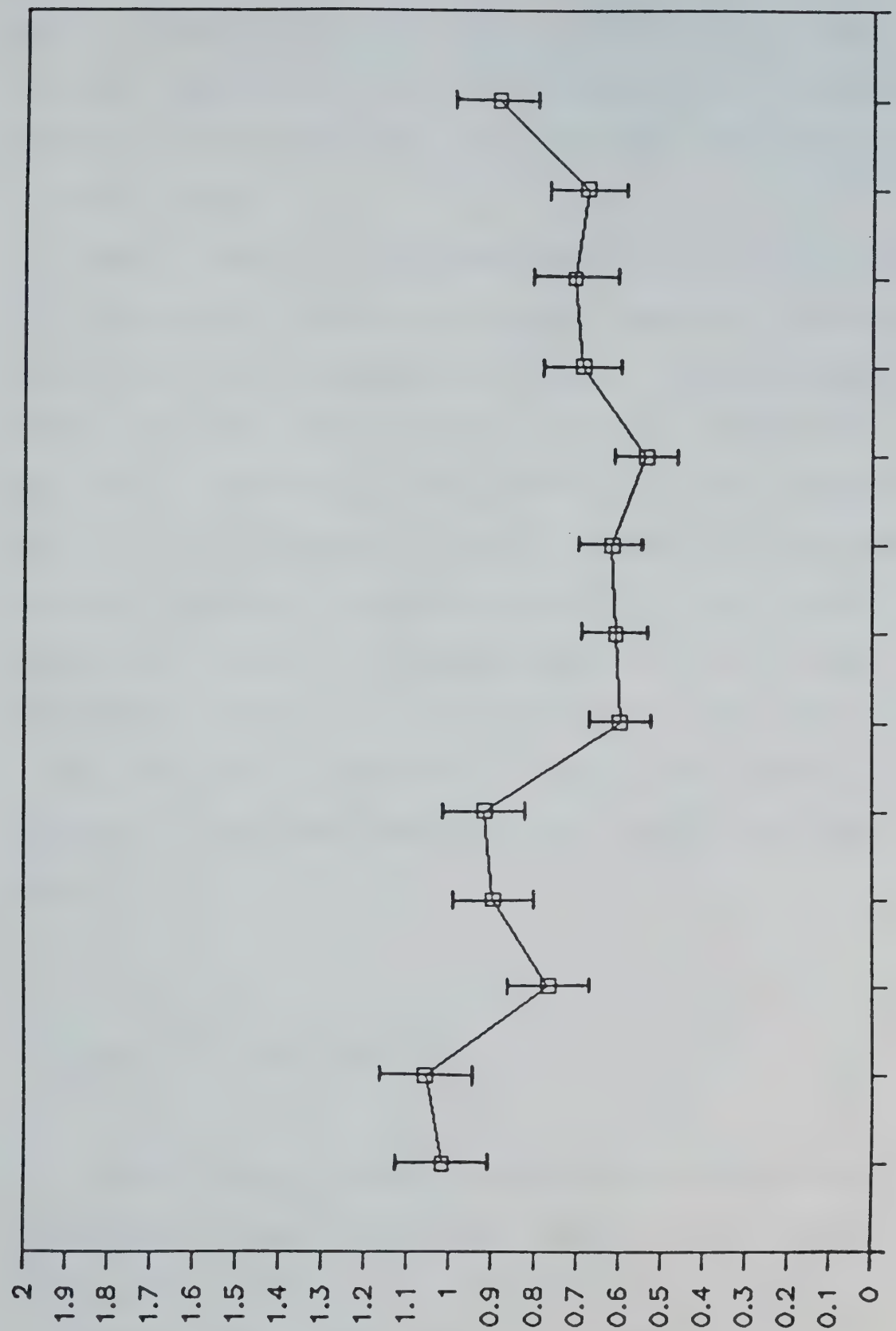

Figure 9: Mean finger pulse wave amplitude responses of both subject groups during the experimental stages.

phases showed a gradual increase back towards rest. However, during the final two stages of the cold phase the mean height of the pulse waves did not increase back to initial levels.

When descriptively comparing the two groups (Figure 10); the young group demonstrated immediate reductions in mean pulse wave amplitude at stage 1 of warm phase 1, cold phase, and warm phase 2. The old group showed less dramatic and rapid reductions in the mean pulse wave heights for each of the equivalent phases. Contrary to the young group's results, the old group showed a continuing reduction in pulse wave amplitude in stages 2 and 3 of the cold phase. During the recovery phase, the finger pulse waves returned to resting values for the younger group, but remained lower than resting pulse heights for the older group.

E. Systolic Blood Pressure

Mean systolic blood pressure measurements during the cold phase were significantly greater than values for all other phases (Figure 11). The mean systolic blood pressure increased from a rest value of 128 mmHg to a value of 132

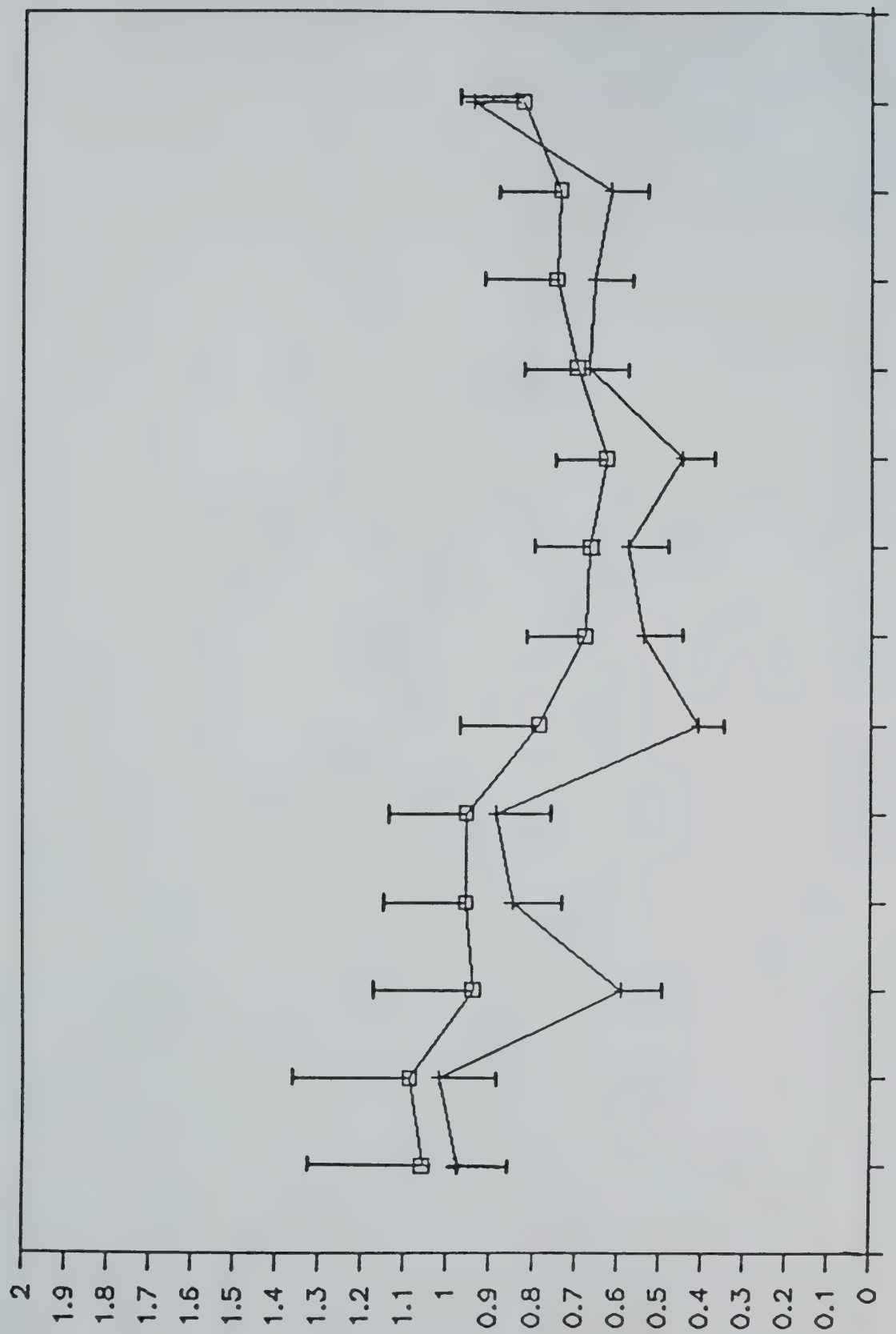

Figure 10: Mean finger pulse wave amplitude responses of the young (+) and old (□) subject groups during the experimental stages.

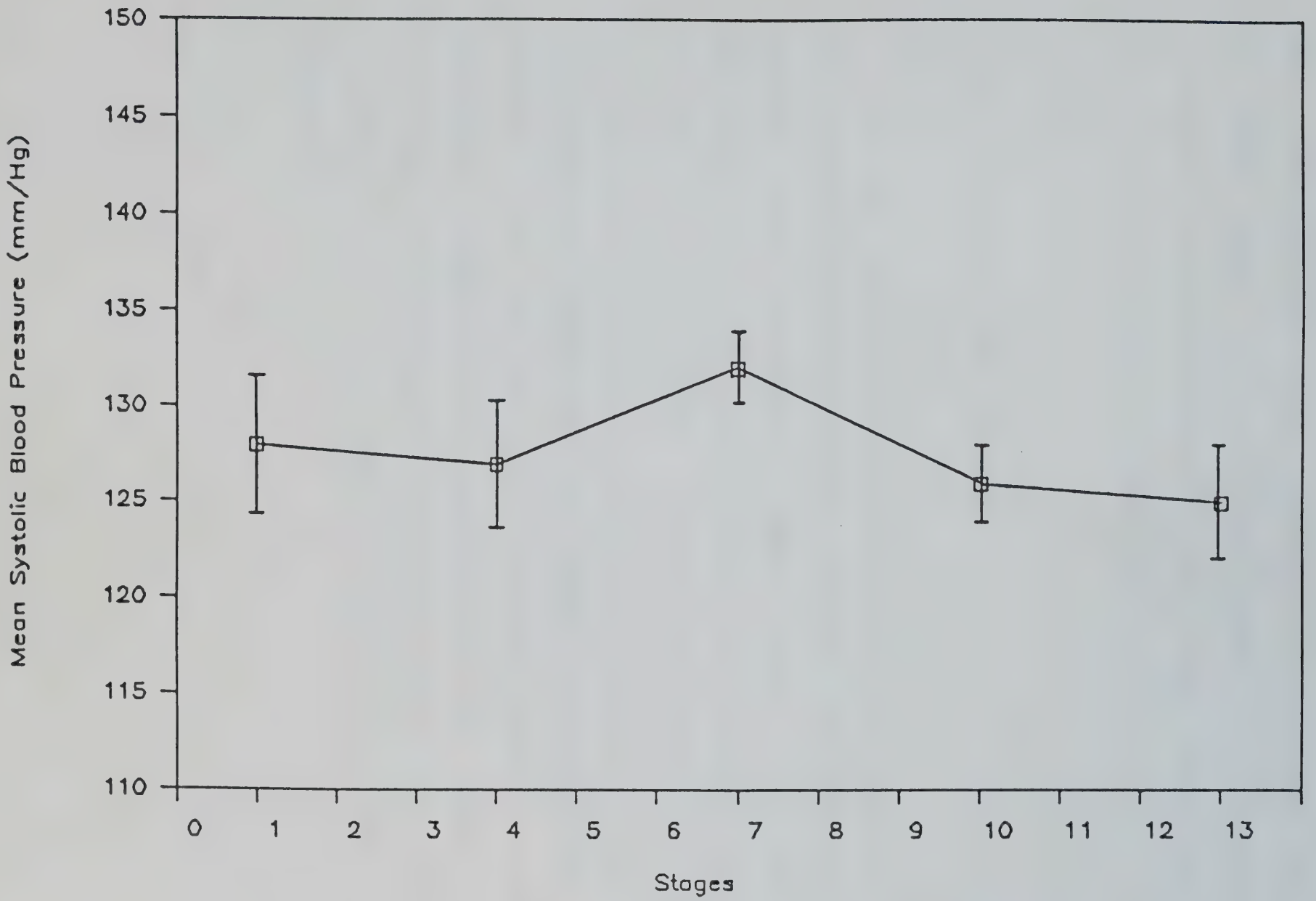

Figure 11: Mean systolic blood pressure responses of both subject groups during the experimental stages.

mmHg during the cold phase. Contrary to the results for the old subject group, the young group showed a slight decrease in systolic blood pressure below rest values during warm phase 1 and warm phase 2 (Figure 12).

F. Mean Diastolic Blood Pressure

The mean diastolic blood pressure for the subjects was significantly higher during the cold phase (88 mmHg) as compared to the rest phase (84 mmHg) (Figure 13). A similar pattern as the systolic blood pressure was observed (Figure 12). The young group showed a slight decrease in diastolic blood pressure from resting conditions during the warm phase and then increased to the highest diastolic blood pressure in the cold phase. The old group showed a gradual increase in diastolic blood pressure from rest values to the highest value during the cold phase, and then a gradual decrease back to resting values in the recovery phase (Figure 14).

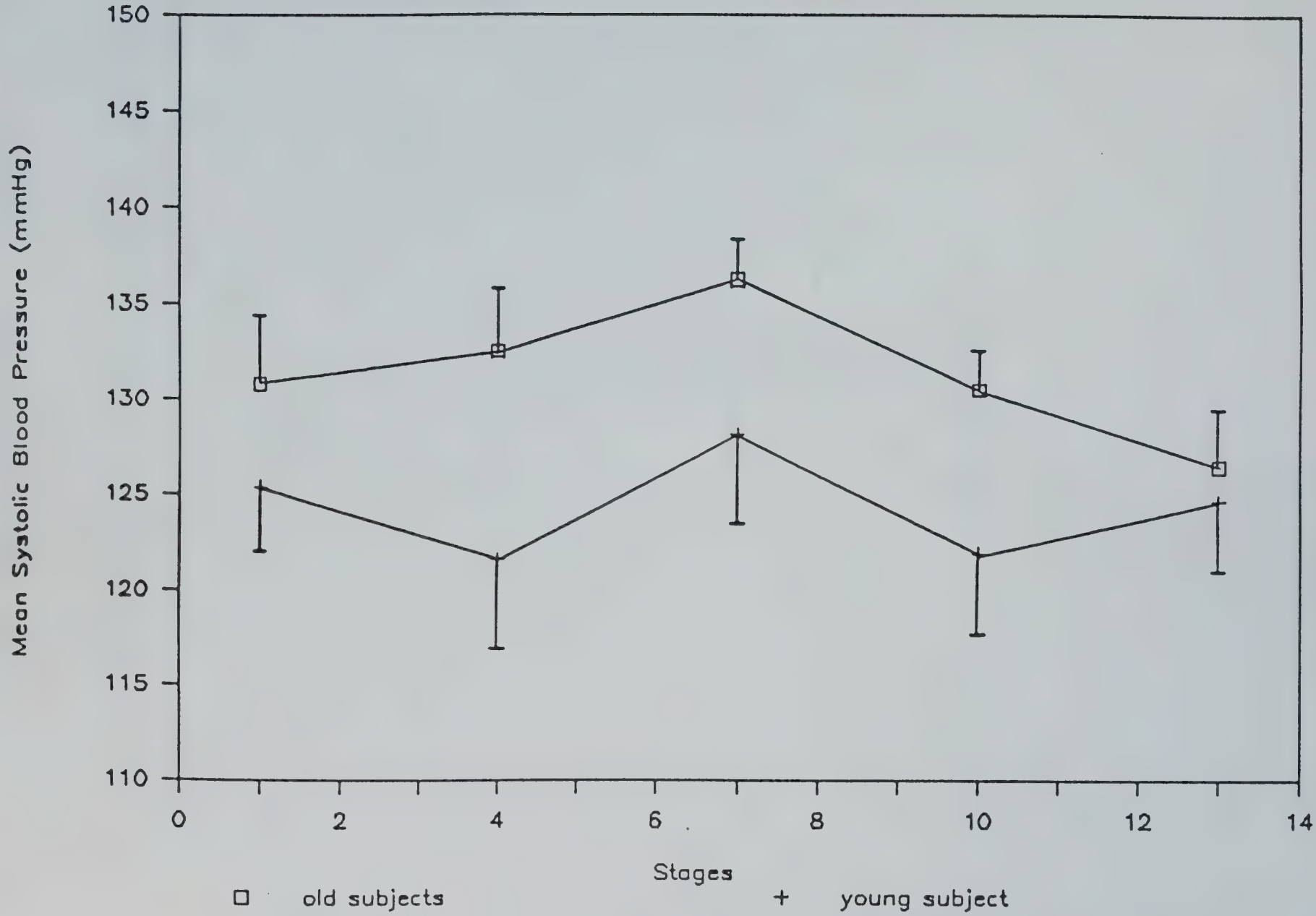

Figure 12: Mean systolic blood pressure responses of the young (+) and old (□) subject groups during the experimental stages.

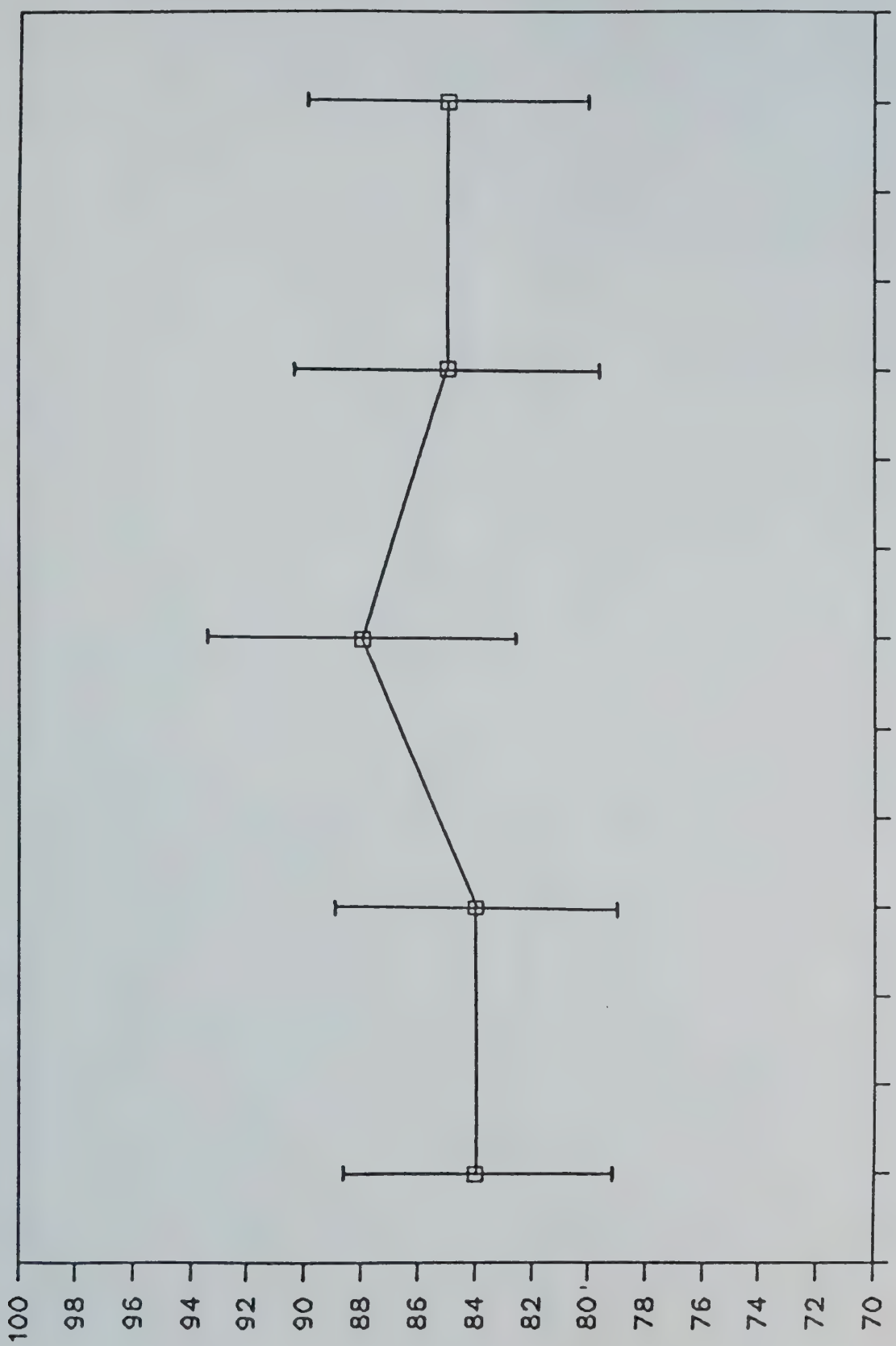

Figure 13: Mean diastolic blood pressure responses of both subject groups during the experimental stages.

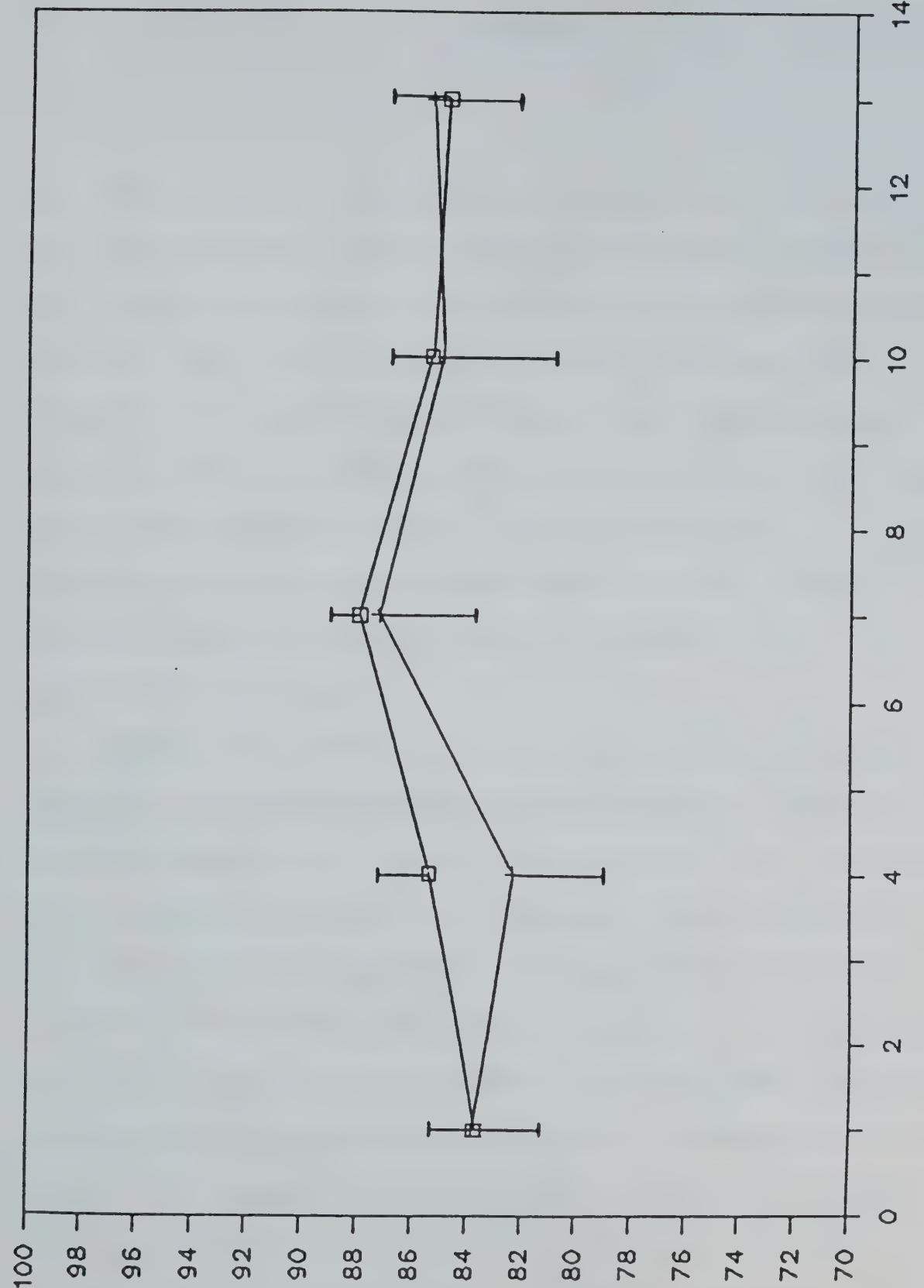

Figure 14: Mean diastolic blood pressure responses of the young (+) and old (□) subject groups during the experimental stages.

5. DISCUSSION

This study utilized a warm wind (20C to 23C) and cold wind (-6C to -8C) with a velocity of 8 m/sec directed at the front of the face in the attempt to elicit the diving response. The characteristics of this response was bradycardia, reduced finger blood flow, and elevated systolic and diastolic bloood pressure. The primary purpose of this investigation was to observe selected cardiovascular variables associated with the diving response and to determine if this response is temperature-dependent.

Bradycardia with the entire multireflex diving response has been demonstrated in many face immersion studies (Magel et al, 1982; Whayne et al, 1971; Stromme et al, 1970; and Irving, 1963). However, there have been very few studies using a cold wind as the stimulus. Leblanc et al (1978) found that both young (20-40 years of age) and old (53-60 years of age) subjects demonstrated significant bradycardia with a cold face stimulus. Hayward et al (1976) showed that heart rate decreased by 9% with a stream of cold air (0 to 10C) directed at the side of the face. The present results showed that a cold wind produces

significant bradycardia which is temperature-dependent. The initial warm wind stimulus did not elicit the bradycardia response in either subject groups. However, the cold wind stimulus elicited a significant bradycardia of 8.2% indicating that the response is temperature-dependent and can not be evoked simply with a wind stimulus. Magel et al (1982) has shown that bradycardia varied in magnitude with water temperatures. This finding has been supported by several other researchers (Parfrey and Sheehan, 1975; Moore, Lin, Laaly, and Hong, 1972a; Kawakami et al, 1967; Whayne and Killip, 1967; and Craig, 1963).

In addition, the present study also demonstrated a significant bradycardia (6.8%) with the second warm wind stimulus. Therefore, the diving response bradycardia was also elicited by a change in stimulus temperature rather than simply by a cold stimulus. This may have resulted from a maintained vagal tone or reduction in sympathetic activity to the heart, from the response to the cold wind stimulus. Although it is the vagal response which produces the reflex bradycardia, it may be that there is also a reduction in sympathetic input to the heart which is masked by the rapid and strong vagal reflex. It is probable that the vagal response is transient, as indicated by an increase in HR in the final two stages of the cold phase,

but the reduced sympathetic activity is maintained for a longer duration. Activation of the vagal inhibition of the heart at the onset of the warm wind stimulus was not challenged by a large sympathetic tone.

Animal studies utilizing trigeminal neurotomy have shown that the afferent pathway of the diving reflex is the trigeminal nerve (Martner et al, 1977; Dykes, 1974; and Andersen, 1963). It has been hypothesized that the trigeminal nerve is also the afferent pathway in humans (Leblanc, 1975; and Parfrey and Sheehan, 1975). This has been supported by human face immersion studies where covering of the trigeminal region of the face caused reduced bradycardia (Stromme et al, 1970). This study supports these results, as significant bradycardia was produced with a wind stimulus directed at the face, while the remainder of the subject's body was covered.

It has been reported that diving bradycardia in man develops gradually with time usually with a latency of 5 to 15 seconds, whereas in the seal submersion of the nose immediately elicits a lengthening of the very first pulse interval (Hauge et al, 1984). The bradycardia response, in both the cold phase and warm phase 2, was rapid in onset occurring within 0 to 10 seconds following the presentation of the stimulus. There was a lengthening of the first or

second R-R interval in 86% of the subjects. These results are in agreement with Stromme et al (1970) who demonstrated that submergence elicits lengthening of the first cardiac interval. Generally it is thought that the abrupt onset of bradycardia is due to inhibition of the heart by the vagus nerve (Dykes, 1974). The utilization of atropine to block the vagal response in both animal studies (Lillo, 1979; Murdaugh et al, 1961; Forster and Nyboer, 1955; Irving et al, 1941; and Richet, 1899) and human studies (Finley et al, 1979; and Heistad et al, 1968) demonstrate an inhibition of the bradycardia response.

The rapid onset of the bradycardia in the present study indicated a vagal response rather than a sympathetic response. The sympathetic response does not appear necessary for development of the bradycardia. This is supported by Finley et al (1979) who demonstrated that bradycardia occurred during face immersion following the administration of alpha (phentolamine) and beta (propranolol) sympathetic blockades.

Most face immersion studies report that the onset of bradycardia occurs immediately upon immersion. The most rapid cardiac retardation occurs between 5 to 15 seconds and maximum bradycardia was reached by 30 seconds of the procedure (Smeland, Owe, and Andersen, 1984; Magel et al, 1982; Stromme et al, 1970; and Whayne and Killip, 1967).

These studies also illustrated that bradycardia is retained for the entire period of facial immersion. Facial cooling studies with a wind stimulus have shown similar results. Hayward et al (1976) found that bradycardia was evident for the whole minute of cold wind stimulus, and Leblanc et al (1978) showed that bradycardia developed wihin 30 seconds after presentation of the stimulus and was maintained for the two minute duration of the cold stimulus.

Similar results were found in this study concerning the rapid onset of bradycardia but not with respect to the maintenance of bradycardia throughout the wind stimulus phases. Contrary to previous facial cooling studies which showed a sustained bradycardia throughout the procedures (Leblanc et al, 1978; and Hayward et al, 1976), this study showed that the final two stages (one-minute and two-minute stages) of the cold phase and warm phase 2, heart rate showed an increase from maximum bradycardia. Bradycardia was not maintained throughout these phases indicating a habituation to the stimulus. The habituation was more pronounced with the warm wind stimulus which showed a rapid increase in heart rate back to resting levels by the one-minute stage. The cold phase had a small increase in heart rate from the maximum bradycardia but these values did not return to resting heart rate values.

Kawakami et al (1967) has suggested that hypercapnia and hypoxia contribute to the maintenance of bradycardia during facial immersion. Finley et al (1979) found that a longer apneic period during face immersion produced an extended bradycardia. It is thought that bradycardia in response to apnea is dependent upon lung volume and intrathoracic pressure (Magel et al, 1982). Since a cold wind protocol does not produce apnea, the subject would not experience hypoxia or hypercapnia, and therefore, would not have changes in lung volume and intrathoracic pressure. The present study also indicates that it is probable that the respiratory stimuli potentiates the bradycardia.

There were no significant differences in heart rate changes over trials between the two subject groups (see Figure 4). However, the younger subjects showed a more pronounced bradycardia than the older subject group. An attenuation of the diving bradycardia with age has been reported previously in normal subjects (Smith and Kampine, 1984; Gooden et al, 1978; and Hong et al, 1970). Younger subjects may have an increased vagal tone which would augment the bradycardia response or may have a decreased sympathetic tone in comparison to older subjects. This increased vagal tone may be due to a higher fitness level in the young group.

Very few studies have investigated the stroke volume and cardiac output parameters of the diving response in humans. Thus, no conclusions have been presented in the literature. Epstein et al (1969) showed that exposure to a cold environment did not significantly change stroke volume values from resting levels, however, this was not with an air stream directed at the face. Contrary to this, Leon et al (1969) found that cold air breathing evoked an increase in stroke volume. Although the present study did not show statistically significant stroke volume changes, stroke volume tended to increase with the presentation of the cold wind stimulus and the second warm wind stimulus. The observed small increases in stroke volume may have compensated for the significant reduction in heart rate which occurred immediately at the beginning of these phases. It could be that stroke volume increments at those phases, would have been greater had the magnitude of the bradycardia been greater.

Stroke volume values between the old and young groups were significantly different, however, there were no statistically significant differences between groups over the trials. A review by Smith and Kampine (1984) reported resting stroke volume values are lower in older subjects due to the effects of aging on circulation. The old group

did show a less pronounced response than the young group but it was not significant. Stroke volume did not increase during the cold phase and warm phase 2 as greatly in the older subjects. Since heart rate reductions were not as pronounced in the older subjects it may be that smaller compensatory stroke volume increments were necessary to maintain a stable cardiac output.

A large reduction in cardiac output in the seal upon submergence has been documented (Elsner and Gooden, 1983) however, this response has not been well established in human facial immersion studies. A recent study investigating the affect of facial immersion during steady-state exercise, reported a decrease in cardiac output by 49% (Bjertnaes, Hauge, Kjekshus, and Soyland, 1984). This indicates that humans are capable of producing similar cardiovascular adjustments to a diving situation as aquatic animals (Bjertnaes et al, 1984). Subjects exposed to a cold environment, demonstrated no significant changes in resting cardiac output values in a neutral environment (Epstein et al, 1969). But, the cardiac output responses to a cold water or cold air stimulus under rest conditions is poorly established.

This study demonstrated no significant changes in cardiac output with any of the wind stimuli. Cardiac output

did tend to decrease slightly, however, with the presentation of the cold wind stimulus. The maintenance of cardiac output throughout the experimental procedures in spite of pronounced bradycardia was due to the simultaneous compensatory increase in stroke volume at those particular stages. This would occur as a result of the Starling effect, where the greater preload increases the force generated by the contracting muscle (Little, 1985). A decreased heart rate will influence stroke volume by allowing for a greater diastolic filling time (Smith and Kampine, 1984). The greater ventricular end-diastolic volume increases the strength of the myocardial contraction. Therefore, stroke volume is increased and cardiac output is maintained despite the significant bradycardia. Under resting conditions a cold wind stimulus at -6C to -8C, does not seem strong enough to evoke a significant reduction in cardiac output as measured by impedance cardiography.

As with stroke volume, cardiac output values were significantly lower in the old group, but showed no significant differences over trials. Lower cardiac ouput values were probably due to the smaller mass of the old group, but also, a diminished cardiac output with age has been reported (Smith and Kampine, 1984). However, the old

group and young group showed an almost identical cardiac output response pattern throughout the experimental procedures. The stroke volume compensatory mechanisms of both groups were effective in maintaining a stable cardiac output.

This study demonstrated qualitative changes in finger blood flow rather than quantitative changes, therefore, it will be discussed in that manner. Each of the wind stimulus phases in this study produced a significant decrease in the mean amplitude of the finger pulse waves indicating a reduction in finger blood flow. Sympathetic vasoconstriction occurring in the periphery, as documented by a reduction in hand and forearm blood flow, has been reported upon presentation of a cold face stimulus (Hayward et al, 1976; Epstein et al, 1969; Leon et al, 1969). This peripheral vasoconstriction evidenced, may have assisted in maintaining the cardiac output by increasing the stroke volume during the reflex bradycardia. The larger venous return, due to the vasoconstrictor response, would contribute to the Starling effect. The increased end-diastolic volume would provide a greater preload, and therefore a stronger myocardial contraction.

Hattenhauer and Neill's study (1975) indicated that this vasoconstrictor response could not be evoked during

warm air breathing, and therefore is a temperature-dependent reflex. It has been suggested that the flow of cold air through the airways is necessary to elicit the vasoconstriction response (Hayward et al, 1976). This study shows evidence that a cold wind stimulus elicits reflex vasoconstriction as indicated by a reduction in finger blood flow. However, a significant decrease in the mean pulse wave amplitude, was also demonstrated in both of the warm wind stimulus procedures. The fact that blood flow was reduced in the first warm wind phase is of more relevance than the blood flow change in the second warm stimulus stage, where the sympathetic tone was already increased due to the previous cold air stimulus. Contrary to previous studies (Hayward et al, 1976; and Wells, Walker, and Hickler, 1960), the sympathetic vasoconstriction response does not appear to be temperature-dependent. It may be that this response is a 'wind reflex' for the conservation of body heat (Ebbecke, 1944). The most dramatic decrease in finger pulse wave amplitude occurred in the cold phase of this study. It may be that the magnitude of the vasoconstriction response is temperature-dependent but the actual elicitation of the response is only wind-dependent.

There were no statistically significant differences

for finger pulse wave amplitude changes between subject groups over the trials. The subject groups showed a similar response pattern throughout the experimental procedures but the younger group showed a more pronounced reduction in finger pulse wave heights in each of the wind stimulus phases. Wagner et al (1974) found that in a cold exposure study finger blood flows were more significantly reduced in the younger subject group. It appears that aging is accompanied by a decrease in peripheral vascular reactivity to cold stress (Wagner et al, 1974). In a neutral environment, older subjects have lower peripheral blood flow values suggesting that aging is accompanied by a decrease in functional cutaneous vascularization (Wagner, Tzankoff, Marino, and Robinson, 1972; and Ring, Kurbatov, Shannon, 1959). The younger subjects in this study showed a greater reduction in finger blood flow which may be due to a greater capability for peripheral vasoconstriction than the older subjects.

Arterial pressure is maintained or increased during the diving response inspite of the significant bradycardia with both face immersion (Stromme et al, 1970; and Leblanc et al, 1975) and upon presentation of a cold wind (Hayward et al, 1976). Face immersion during exercise produced a 69% increase in mean arterial systemic blood pressure in a

study by Bjertnaes et al (1984). In several cold air breathing studies, mean arterial pressure and systolic blood pressure have increased significantly from resting levels (Hattenhauer and Neill, 1975; Neill et al, 1973; and Leon et al, 1969). The mean systolic blood pressure during the cold phase in this study was significantly higher than all other phases of the experimental procedures. Mean diastolic blood pressure showed the same response pattern, the cold phase value was significantly greater than the rest phase value. The increased systolic blood pressure occurred in spite of the significant bradycardia occurring simultaneously at this cold phase, indicating a strong vasoconstriction response. The moderate increases in stroke volume at these stages may be a contributing factor in elevation of systolic blood pressure. It can be concluded that the vasoconstriction response outweighed cardiac output reductions, causing an augmentation of mean systolic blood pressure. Systolic and diastolic blood pressure were not significantly increased during the warm wind phases. However, some vasoconstriction occurred since arterial blood pressure was maintained in spite of the evident bradycardia.

There were no significant differences between the subject groups for systolic blood pressure values, however,

the older group demonstrated higher systolic blood pressure values throughout the procedures. An increased peripheral resistance with aging, produces higher arterial blood pressure values in older subjects (Smith and Kampine, 1984). The sympathetic response to a cold wind stimulus seems to be equivalent for both a young and old population.

SUMMARY

This study demonstrated that a cold wind (-6C to -8C) at a velocity of 8 mm/sec can elicit the diving reflex in both young and old subjects. Subjects exhibited significant bradycardia, peripheral vasoconstriction, increased arterial blood pressure. Also, there was a moderate increase in stroke volume. Hauge et al (1984) reported that man's bradycardia response is different from diving animals because bradycardia develops gradually and becomes more intensified throughout the immersion procedure. A cold wind stimulus elicited an immediate onset of bradycardia in this study, 86% of the subjects showing a lengthening of the first or second pulse interval. This bradycardia was not

sustained as in many face immersion studies (Smeland et al, 1984; Magel et al, 1982; Stromme et al, 1970; and Whayne and Killip, 1967). It is probable that this was due to the absence of respiratory stimuli (changes in lung volume and intrathoracic pressure) which are responsible for the potentiation of the bradycardia response.

When the face was exposed to a cold wind there was a significant increase in arterial blood pressure indicating increased sympathetic activity, but the concomitant bradycardia illustrated an enhanced vagal activity. It appears that the diving response, whether elicited by a water or wind stimulus, is temperature-dependent with the exception of the peripheral vasoconstriction response which may be a "wind reflex". Although, not statistically significant the young group demonstrated a more pronounced diving response. The elicitation of the diving response under the conditions of this study has implications for individuals who experience angina pectoris.

IMPLICATIONS

The diving response does have implications for the development of angina pectoris in coronary disease patients. This study simulated outdoor conditions where only the face was exposed to a cold wind with the remainder of the body covered. The diving response can be elicited in both young and old people under these conditions. A significant bradycardia, increased systemic arterial blood pressure, and peripheral vasoconstriction was observed. For patients with coronary disease this increased arterial pressure, although occurring concomitantly with the reflex bradycardia, may produce additional pressure work for the heart thus evoking angina pectoris.

It is hypothesized that the increase in arterial pressure causes greater pressure work for the heart. The net pressure-volume work of the heart is a product of ventricular pressure and the stroke volume, over the period of systole (Little, 1985). This study provided evidence that a cold wind produces a significantly elevated arterial blood pressure. Additional pressure work occurs when the heart continues to pump a normal stroke volume against a higher than normal arterial pressure (Little, 1985). The simultaneous reflex bradycardia, demonstrated in the

present study, produced a small increase in stroke volume to maintain cardiac output. If the heart must move a larger than normal stroke volume against an elevated arterial pressure, this adds to both the pressure and volume work of the heart. The increased net pressure-volume work would be directly related to the myocardial oxygen demand. The increase in myocardial oxygen requirements, evoked by a cold stimulus, would create an imbalance in myocardial oxygen delivery and oxygen demand in areas of the myocardium to which oxygen delivery is restricted by coronary vascular disease (Epstein et al, 1969). This supports the theory that the elicitation of angina pectoris with cold exposure, is due to the increased oxygen demand on the heart, as a result of the additional pressure-volume work.

Some investigators have hypothesized that cold stimulation of the face produces angina pectoris due to reflex constriction of the coronary arteries rather than as a result of increased myocardial oxygen demand (Sorensen, 1966; Berman and McGuire, 1950; and Gilbert, Fenn, LeRoy, and Hobbs, 1941). However Hattenhauer and Neill (1975) found no detectable constriction of coronary arteries visualized arteriographically in coronary patients. Of the 17 patients in that study, 4 patients developed angina

pectoris, and showed electrocardiographic and chemical signs of hypoxia. It was suggested that cold air inhalation constricts minute coronary vessels affecting blood flow in potentially ischemic areas of the myocardium (Hattenhauer and Neill, 1975).

It may be that angina pectoris produced from a cold wind is due to coronary spasm, however, the present study does indicate that myocardial oxygen demand is increased as a result of a cold wind stimulus even under resting conditions.

FURTHER STUDY

Further research is necessary to better understand the implications of the diving response. A similar facial cooling study using a moderate exercise protocol would illustrate the exercise response modifications due to the diving reflex. Therefore, cardiovascular adjustments while performing physical activity in the cold would be identified. This investigation would be relevant in determining the etiology of angina pectoris since it often occurs during some level of physical exertion.

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APPENDIX 1

FACIAL COOLING STUDY

CONSENT FOR TESTING

I, _____

(please print)

authorize Aaron Tubman to administer a facial cooling test for the purpose of collecting experimental data. I understand that I may discontinue the test if at anytime I experience unusual discomfort. I understand that I will undergo the procedures outlined below and I have the opportunity to question and discuss the exact procedures that will be followed.

Procedure to be followed:

1. Electrocardiogram recording - requires the placement of 4 circular, adhesive electrodes on the chest and back. Shaving of these areas may be necessary for some subjects and the electrodes may cause some skin irritation or redness.

2. Stroke volume measurements - requires the placement of 4 tape electrodes; 2 electrodes around the neck and 2 electrodes around the abdomen. These electrodes may cause some skin irritation or redness.

3. Finger blood flow measurement - requires the placement of an electrode around the thumb.

4. Facial stimulus - the facial region will be exposed to a cold and warm air stimulus. This wind stimulus may cause tearing or slight eye irritation.

5. Blood sample - a small blood sample will be taken using the finger prick method to determine hematocrit.

I acknowledge that I have read this form and I understand the test procedures to be performed and the inherent risks and I consent to participate. I understand that the data will be used for research purposes in a form that will not allow personal identification.

DATE:_____ SIGNATURE:_____

APPENDIX 2

TABLE 2

Old Group Mean Data

ACTIVITY	Stage #	HR (b/min.)	SV (ml/b.)	CO (l/min.)	SBP (mmHg)	DBP (mmHg)	MAP (mmHg)	Finger bd flow
Rest	1.00	70.00	75.20	5.29	131.00	84.00	99.00	1.06
Rest	2.00	71.00	77.20	5.47				1.09
S. Warm	3.00	70.00	79.00	5.49				0.94
Warm	4.00	71.00	76.60	5.44	133.00	85.00	101.00	0.96
E. Warm	5.00	72.00	75.30	5.44				0.96
S. Cold	6.00	67.00	76.40	5.12				0.79
Cold	7.00	69.00	77.50	5.37	136.00	88.00	104.00	0.68
E. Cold	8.00	70.00	74.30	5.18				0.67
S. Warm	9.00	67.00	77.50	5.21				0.63
Warm	10.00	71.00	72.80	5.19	131.00	85.00	100.00	0.70
E. Warm	11.00	71.00	73.00	5.15				0.75
Recovery	12.00	69.00	75.50	5.17				0.74
Recovery	13.00	71.00	75.00	5.30	127.00	85.00	99.00	0.83

TABLE 3

Young Group Mean Data

ACTIVITY	Stage #	HR (b/min.)	SV (ml/b.)	CO (l/min.)	SBP (mmHg)	DBP (mmHg)	MAP (mmHg)	Finger bd flow (mm)
Rest	1.00	75.00	98.00	7.23	125.00	84.00	98.00	0.98
Rest	2.00	74.00	98.10	7.16				1.02
S. Warm	3.00	73.00	99.30	7.14				0.59
Warm	4.00	74.00	97.30	7.11	122.00	82.00	95.00	0.85
E. Warm	5.00	74.00	97.20	7.14				0.89
S. Cold	6.00	67.00	104.30	6.86				0.41
Cold	7.00	72.00	97.40	6.89	128.00	87.00	101.00	0.54
E. Cold	8.00	72.00	98.80	6.96				0.58
S. Warm	9.00	69.00	103.80	7.05				0.45
Warm	10.00	75.00	96.70	7.10	122.00	85.00	97.00	0.67
E. Warm	11.00	75.00	94.90	7.00				0.66
Recovery	12.00	76.00	97.10	7.18				0.62
Recovery	13.00	77.00	95.10	7.14	125.00	85.00	99.00	0.95

TABLE 4

Old and Young Groups' Mean Data

ACTIVITY	Stage #	HR (b/min)	SV (ml/b.)	CO (l/min)	SBP (mmHg)	DBP (mmHg)	MAP (mmHg)	Finger Bl.Flow
Rest	1	73	86.59	6.26	128	84	99	1.02
Rest	2	73	87.65	6.32				1.06
S. Warm	3	72	89.12	6.31				0.77
Warm	4	73	86.93	6.27	127	84	98	0.9
E. Warm	5	73	86.26	6.29				0.92
S. Cold	6	67	90.34	5.99				0.6
Cold	7	70	87.55	6.13	132	88	103	0.61
E. Cold	8	71	86.55	6.07				0.62
S. Warm	9	68	90.66	6.13				0.54
Warm	10	73	84.74	6.15	126	85	99	0.69
E. Warm	11	73	83.92	6.08				0.71
Recovery	12	72	86.28	6.18				0.68
Recovery	13	74	85.06	6.22	125	85	98	0.89